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A STUDY OF CHILD CARE IN THE FIRST TWO YEARS OF LIFE

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THE following study was carried on for two reasons: First, contact over a number of years with the Well Baby Conferences, organized by the Visiting Nurse Association in various parts of New Haven, suggested the fact that a psychologist in attendance, as well as a pediatrician, would be of service to the mothers in stressing additional aspects of the child's growth, such as his mental development, the necessity for the formation of regular habits, and early consideration of the behavior expressive of emotional states. The second purpose of the study was to determine the problems of management actually encountered by parents in the first two years of a child's life. It was thought that by observing the same children over a period of two or three years it might be determined whether atypical behavior which assumed importance in the mind of the mother or other observers in the early months was followed by marked behavior deviations in the later months.

A Well Baby Conference in one of the suburbs of New Haven was available in the autumn of 1928, owing to the interest and cooperation of the Hamden Visiting Nurse Association.* This conference was an excellent one for the project, first because its enrollment was small enough to make fairly intensive study of each child a possibility, from both the pediatric and the psychologic points of view; second, the parents were average or slightly above with respect to economic status, educational opportunity, and attitude toward child care.

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*We wish to express our appreciation to the members of the staff of the organization for their cooperation and generous expenditure of time in behalf of the study.

Weekly conferences were attended by the pediatrician and the psychologist from the autumn of 1928 until the spring of 1930. Follow-up visits were made in the spring of 1931. The procedure usually followed in such Well Baby Conferences was used by the pediatrician. She also studied the infants at home or at the Clinic of Child Development from the developmental and behavior aspects when they were four, six, eight, and twelve weeks of age.

Opportunity to have a developmental psychologic examination was offered to the mother of every child under two years of age whose name appeared on the conference list. The mothers were informed that the psychologist's chief interest lay in the development of the baby's mind, rather than his body, and in the way in which his habits were being formed. Children who were under six months of age at the time of the first contact with the psychologist (hereafter referred to as Group I) were taken to the Clinic of Child Development for detailed observation of the child and interview with the mother. These detailed studies were made at lunar month intervals (four weeks) during the child's first year. Follow-up studies were made in less detailed fashion at intervals of three months during the second year, and six months or yearly following the second birthday. Children who were over six months of age at the time of the first contact (Group II) were studied less intensively with the same intervals between observations. A quiet room adjoining the pediatrician's was available for these less intensive studies.

The results of the examination were summarized briefly for the mother each time. Remedial suggestions regarding the child's behavior were given very sparingly for two reasons: first, because part of the plan was to record the methods used by the mothers themselves in meeting behavior difficulties; second, because it was believed that in certain instances the difficulties would be increased by calling attention to them, while they might pass if unmentioned. This proved to be the case often enough to justify the theory. Persistent difficulties were of course discussed fully with a view to finding a way out. The detailed interviews with the mothers covered the following points:

A. Feeding:

Hours, foods (actual amounts).
Time consumed at each feeding.
Behavior (distractibility, speed, methods of rejecting, etc.).
Variability of appetite.

B. Sleeping:

Hours.
Regularity.
Quality of sleep (nights disturbed by internal stimuli, etc.).
Effect on sleep of external disturbances (noise, being moved, etc.).

Behavior when "put down" (special measures to induce sleep).
1. At accustomed time and place.
2. Under changed conditions.
Behavior on waking.
Effect of unusual day on sleep (upset schedule, unusual social stimulation).

C. Dressing:
Bathing behavior.
Changing.
Number of times daily,
Pre- and postchanging behavior,
Training measures employed.
Undressing at night, rubbing, etc.

D. Play (alone or with others):
Number of persons playing with the child. Type of relationship.
Objects used in play.

E. Expressive behavior and personality traits:
Crying
Explained,
Unexplained,
Time of day.
How is baby quieted?
Laughing
Incidence,
Stimuli.
Moods (sensitiveness to mother's moods; duration; changeableness).
Demonstrativeness of or responsiveness of baby.
Vocalization.
Fears.
Anger.
Reaction to "discipline."
Habits (thumb, pacifier, rocking, handling of contact type).

F. Behavior observations during time at clinic.

G. Suggestions made to the mother in the course of the interview.

The same general headings were used for the shorter interviews. In studying the second group, details were asked for only when the mother reported difficulty in one behavior field or another.

Following each interview the facts were summarized on a card which was placed in a file on the pediatrician's desk. By turning to these cards when a child was brought to her, she was enabled to discuss with the mother in the interval between the periodic psychologic examinations, any difficulties which were found recorded there. When questions regarding training were put to the pediatrician, she in turn made note of her suggestions for the psychologist's reference. Special difficulties were discussed by the pediatrician and psychologist following the conference in order that agreement with respect to suggestions might be reached.

Fifty-nine children were studied, twenty of them intensively. Thirteen children were seen only once; the others, two to eleven times.

The average number of interviews per child was five. The numbers seen at the various age levels are given in Table I.

TABLE I

AGE LEVEL OF INTERVIEWS AND NUMBER OF CHILDREN AT EACH AGE LEVEL

	WEEKS														MONTHS									TOTAL
Age levels	4	6	8	12	16	20	24	28	32	36	40	44	48	52	15	18	21	24	27	30	36	48+		
Number of children	5	8	17	15	11	9	9	9	12	18	9	11	6	15	16	15	11	13	5	5	10	6		235

It may be said of those mothers who kept their appointments, that the larger proportion were very much interested. It is probable that for many the pleasure of having an appointment and an "outing" were real factors. Many young mothers are very much isolated in their households with their babies, and any opportunity to discuss their "job" is a real relief and pleasure. (One young mother in another research group said, "It's heavenly to find some one who is paid to listen to me talk about the baby.")

In spite of the fact that they were interested and cooperative, very few of the women spontaneously asked questions or sought suggestions concerning the psychologic implications of training measures. It was obvious that their preparation for the care of their children had not led them into this field of thought. Only one or two asked about books; probably some of them listened to daily talks on child care over the radio.

The children studied were rather better than the average with respect to mental development. The mothers were as much interested to "check up" on the increment of mental growth as on that of physical growth, enjoying the assurance that good progress had been made in the interval between the examinations.

In the first group, 50 per cent were first children. Of the second group, 33 per cent were the first children in the family, and 16 per cent were members of families in which there were five or more children.

Of the first group, the pediatrician and psychologist, making the decision independently, agreed in the opinion that seventeen were essentially stable individuals. One child was probably stable, but because of faulty handling behaved as many unstable individuals do. Two children presented definite evidence of instability. Further contact with these individuals will be of the greatest interest. In the second group twenty-seven of the children were characterized as stable. It was questionable whether eight of the others were essentially unstable or whether again faulty handling were not responsible for the highly expressive, irritable behavior. In this group one individual only was characterized as unstable.

Behavior in the various fields (feeding, sleeping, play, etc.) is discussed in that part of the report which follows. The study lacks accuracy in that all hours were recorded simply as they were reported by the mothers rather than from actual observation. In many instances the report must have represented the standard toward which the mother was striving rather than the actual state of affairs.

The fact of most interest to us was that infants comparatively often deviated from the ideal schedules laid down in books on infant care with no observable harmful effects. It would seem to be true that in writing such books far too little emphasis has been laid on individual differences. No matter in what manner a mother acquires her knowledge of an ideal schedule, whether from books, neighbors, or professional workers, the fact that her infant deviates from it is often a cause of anxiety. For example, not all the infants eat or can eat the same amount of food, but many mothers worry if the standard eight ounces of milk or prescribed cereal is not consumed. Possibly here the seeds of many feeding difficulties are sown, as a tense effort is made to get the child to eat the food that has been made to seem desirable. With a weight chart at hand which showed a steady gain, one could assure the mother of a child with small food requirements that everything was as it should be, with resulting beneficial effect on her attitude at his mealtimes. Probably the major function of both pediatrician and psychologist in these and similar situations was to reassure concerning the child's well-being.

DISCUSSION OF INFANTS' BEHAVIOR AS REPORTED BY THE MOTHERS

For the age period of four to twelve weeks, the infants' sleeping and eating are so peculiarly interdependent that they are considered together in the following paragraphs.

In hospital nurseries, normal infants sleep almost continuously except during periods of bathing and feeding, including frequently ten or fifteen minutes before feeding. Prolonged crying usually indicates hunger which can readily be counteracted with supplementary feedings. By the time the child leaves the hospital his food requirements have been satisfactorily met, he is gaining in weight, and in every way seems well adjusted.

Under favorable circumstances this good adjustment continues at home, but all too frequently the posthospital period marks the onset of trouble for both mother and child. Rather suddenly the mother resumes her household duties, adding to them the anxious responsibility of caring for a new baby. This would prove a fatiguing undertaking for the healthiest individual, but with the mother still physically depleted and emotionally tense and unstable, it is small wonder that

her breast milk is liable to be affected quantitatively if not qualitatively and that immediately thereafter the baby becomes hungry, fussy and "colicky."

Accordingly it was not surprising to find four of the six babies whom we visited at four weeks crying too much, sleeping too little, in general poorly adjusted, and their mothers in constant need of reassurance and advice, particularly in regard to feedings.

The technic of feeding the child presented no problem. The infant's appetites were consistently good; breast milk and formulas were alike well taken. But the babies were hungry, and insufficient or improper food appeared as the outstanding cause not only for the few digestive disturbances but indirectly for the infants' marked restlessness. Whether this restlessness was increased by the direct communication of the mother's emotional tension and uncertainty as she handled the child is a subject for conjecture only.

The most sensitive and reliable indicator of the adequacy of the régime planned for these infants' early adjustment, especially from the nutritional standpoint, was found in their sleep curve. The percentage of infants whose total sleep approximated the expected or minimum ideal was only 33 per cent at four weeks, but increased steadily to 60 per cent at twelve weeks. Only at sixteen weeks did it first seem fair to us to regard the actual hours of sleep recorded as approximating a normal average. Striking also were the wide variations in the total hours of sleep not only from child to child at a given age level, but from week to week in a given child. Again it was not until sixteen weeks or later that the individual child's hours of sleep became habitual.

A. Feeding: Table II summarizes the findings with respect to type and times of feeding. The majority of children were fed five or six times daily during the first year and a fairly large proportion of them through the second year. When milk was given as much as half an hour before or after other food, it was counted as a separate feeding. Many of the women were successful in getting the children to take larger amounts of food if a short interval was allowed between the solids and the milk, a practice always advised against.

More difficulties were reported in regard to feeding than in any of the other behavior fields, though at least half of all the children seen at every age level were reported to eat well and the difficulties were usually minor ones. In many instances difficulties reported at one interview had been forgotten by the next. Only one of the twenty children intensively studied need be characterized as a feeding problem. Three of the thirty-six less intensively studied children presented real difficulties.

During the first year of life, although appetite was reported constant in about 50 per cent of all the children studied at each age level, certain variations were also reported such as marked impatience before feedings or appetite which varied from day to day or meal to meal.

One to three children at every age level up to one year were reported to like everything, but many dislikes were also reported. Water, new foods, vegetables, and cod liver oil were the most frequently mentioned. Solids were occasionally refused but taken if made a little thinner. Objection had therefore been registered to the consistency rather than to the taste. On the other hand, it was sometimes found that after solids had been offered to the child he preferred them to liquids. To these children milk was offered before solids at mealtimes; while to those who continued to prefer their bottles to vegetables or other solids, the solids were offered while the

TABLE II

SUMMARY OF FEEDING DATA

	WEEKS									MONTHS			
Age level	16	20	24	28	32	36	40	44	48	52	15	18	21	24
No. of children	10	9	8	9	12	18	9	12	6	12	15	14	11	9
Breast fed	60%	55%	37.5%	22%	25%	28%	11%	0	0	0				
Breast fed with supplementary	10%	13%	12.5%	0	0	6%	11%	16%	0	8%				
Formula only	30%	22%	50 %	78%	75%	66%	78%	84%	100%	92%				
Three-hour schedule	1	1		0	0	0	1			1?				
Four-hour schedule	8	8	8	8	12	9	6	7	3	1				
No. fed twice during night	4	3	0	0	0	1	0	0		0				
No. fed irregularly	1			1		9	2	5	3	10	4	3		4

child was most hungry. Only once did a mother report that the child refused a food disliked by herself, while two mothers expressed surprise because their babies had not refused a food which they disliked.

The children had various methods of registering their objections. The spoon filled with the disliked food was beaten off with the child's hand. Mouths were closed and heads turned away. Milk was passively refused by cessation of sucking. Occasionally feeding times were difficult because the children were so active that they could not be kept quiet even while they were fed, or a child was very easily distracted and would not eat if there were other people in the room.

The mothers met their difficulties in a variety of ways. The child was amused while eating in order to ease the situation. If milk was refused it was offered again and again until it was accepted regardless of what this did to the schedule. Children who were well able to hold their bottles were helped because they then took more. Although the usual method was to persevere in giving the child the food refused,

occasionally the mothers accepted the child's negative as valid and allowed the child's diet to become restricted to what he took easily. Certain difficulties were due simply to the fact that the child had not acquired the technic for using the new implement, bottle, cup, or spoon, and were obviated by a little practice. Each time that the pediatrician introduced solids into the child's diet the mechanical difficulty for the child in manipulating solids was explained and the mother was told how to help him. Other variations were accepted by

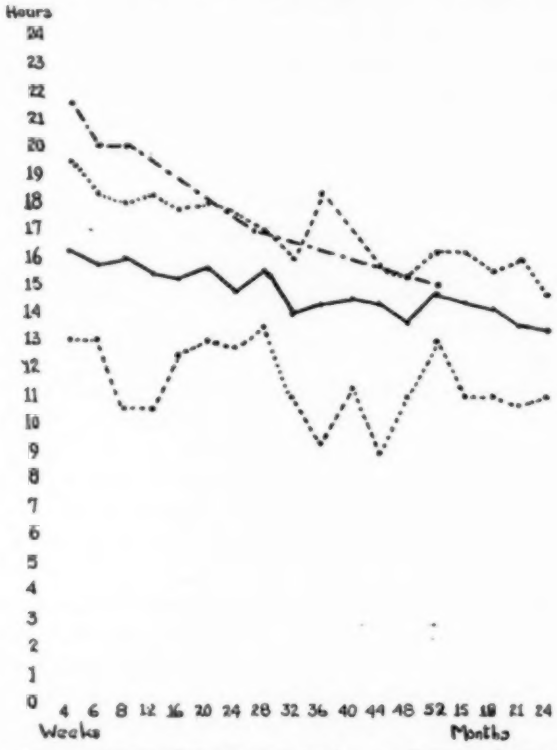

Fig. 1.——Average hours' sleep per age level. . . . Upper and lower limits of variation. —.—. Average hours' sleep recommended by the U. S. Children's Bureau up to one year.

the mothers as due to physical condition, poor appetite coincident with teething, upper respiratory infection, or other illness.

The fact of interest was, however, that in the second year the majority of the children of all age levels were reported to be taking all foods well. Out of fifty interviews it was reported four times that the children's food requirements were small but constant. Occasionally certain foods were vehemently refused but only once was a new food refused.

B. Sleep: Marked individual differences with respect to actual hours of sleep were vividly brought out. Apparently infants differ as much as adults with respect to the amount and quality of their sleep. For the most part, after the initial adjustment to the conditions of living was made, infants who slept comparatively little in the early months, slept consistently a little less as the year went on. Even such children gained weight satisfactorily, however.

The graph in Fig. 1 represents what was learned concerning the hours of sleep in the first two years of life. As might be expected, the hours of sleep are progressively less, with marked variations at one or two age levels. These variations are to be accounted for by the necessary inclusion of one or two very good or very bad sleepers in a

TABLE III

PERCENTAGE OF CHILDREN SLEEPING AT REGULAR HOURS

	WEEKS										MONTHS			
Age levels	16	20	24	28	32	36	40	44	48	52	15	18	21	24
No. of children	11	8	8	9	13	18	9	11	5	17	14	15	10	12
Percentage of children sleeping regular hours at night	81.8	75.0	87.5	88.8	76.9	72.2	88.0	90.9	80.0	76.4	92.8	100	80.0	100
Percentage of children sleeping regular hours during the day	81.8	62.5	62.5	77.7	69.2	72.2	33.0	81.8	40.0	64.7	78.6	80.0	70.0	83.3
Percentage of children sleeping through the night	33	33	50	55	50	55	87	75	83	72	93	93	100	100

small number of cases. The fact is brought out that the average number of hours of sleep per child during the first two years was always less than the amount recommended as desirable in the publications of the United States Children's Bureau.* In their pamphlet entitled *Infant Care* it is suggested that children should sleep twenty to twenty-two hours per day in the early weeks of life, and fifteen hours per day at the end of the first year—a difference of five to seven hours. Our figures indicate an actual average of sixteen to seventeen hours in the early weeks, and thirteen to fourteen hours at the end of the second year—a difference of two to three hours. Our upper limit of variation approaches the Children's Bureau recommendations in the early months and exceeds it in the later months. Possibly it is a good policy to hold an ideal before mothers, but it causes much anxiety to

*Infant Care Publication No. 8 U. S. Department of Labor, Children's Bureau 1929. Later editions emphasize individual differences.

those mothers whose children fall most short of it. Our study would indicate the necessity for determining with greater accuracy, and stressing as well, individual variations within limits that do not threaten the health of the child.

Once the infants and mothers had settled down into an habitual schedule, sleep, as reported, was very regular. Table III shows the percentage of children who slept each day at regular hours. It was of interest to find that a comparatively large percentage slept through the night even in the early months and that no night wakings were reported at two years of age.

As one might expect, regularity was less often achieved during the day than during the night. There was no marked improvement in regularity in the course of two years. Daytime regularity apparently depended somewhat on the child's organism and his rhythmic need of sleep. In certain instances the infant was not "put down" during the day unless he seemed sleepy.

Habitual forms of behavior at sleeping times, such as thumb-sucking or desire for the bottle, were watched with interest. In only one or two instances was such behavior continued into the second year.

C. Bathing and Dressing: Even at the age of four weeks it was usually reported that the baby "just loved his bath." The warm tub brought a conspicuous quieting in very young infants which only later developed into more active expressions of enjoyment. Indeed this sudden subsidence of facial and bodily activity when held in warm water, the maintenance of an immobile position in which the extremities were flexed and adducted, was so striking that one could not but wonder whether the water in its resemblance to amniotic fluid did not set off a reflex or general behavior pattern conditioned in utero. Removal from the tub marked the resumption of squirming and kicking and in a few instances occasioned sharp crying usually attributed to the sudden stimulus of cool air.

As soon as the mother had acquired her technic, she as well as the child enjoyed the bathing period. Indeed the tub was so much enjoyed that after twenty-four weeks one to four children at every age level registered protest when they were removed from it. The infant often protested during the cleaning of the ears, nose, eyes, and mouth, but this had dropped out by the end of the first year. Up to one year it was occasionally reported that the baby was fatigued or overhungry before the end of the bathing period so that he was fussy. Bonnets or clothes which must be pulled on over the head were frequently disliked. The activity of certain children at dressing time made their management an ordeal.

Without doubt the mother's attitude was very important in influencing the children's behavior at this time. There is a certain inevitability about the things that must be done when bathing or dress-

ing a baby. Whatever his protest the mother must persevere once she has begun. Furthermore, there is no cause for fundamental anxiety as in eating, sleeping, and elimination situations. The bath is undoubtedly enjoyed and the infant appears to learn that the rest of the process will be put through regardless of his protest. It is unfortunate that the same attitude cannot be simulated in other care situations.

D. Elimination: Training in control of the bowel movement usually began before that for bladder control, undoubtedly because defecation occurred only once or twice during the day and usually rhythmically. Mothers seemed to be divided about evenly when it came to deciding whether it was more work to wash diapers or frequently go through the process of placing the young infant on a nursery chair. Training was usually begun before the end of the first year.

Sixteen children were reported to have movements at regular hours; nine at irregular hours; four were said to vary between regularity and irregularity. (This item was recorded in only a few instances in the second group.)

The women found that the best times during the day to begin training for bladder control were just after a feeding as the infant often voided a few minutes after finishing a nursing or a bottle and, if dry on waking, immediately following a nap.

E. Play: Babies of four weeks seldom show activity worthy of the name of play. If well fed and comfortable and left to their own devices they soon fall asleep. By six weeks, however, their interest in themselves and in their immediate surroundings has clearly emerged. In our series, six of the eight babies six weeks of age were content at certain hours of the day to amuse themselves by their own gentle activity and the first forms of smiling and vocalization. These periods were reported to last from one-half to two hours and occurred particularly before or after the 6 A.M. feeding. Later in the day attention from members of the family seemed necessary to prevent fussing.

The waking hours of even young infants can become a burden if the mother's attitude with regard to them is not a casual, serene one. It is on the whole encouraging to find that an average of 54.5 per cent of the children during their first year were capable of providing interest and amusement for themselves when alone (crib, carriage, room, outdoors); and 56.2 per cent during the second year.

Taking the average of all those reported upon, 45 per cent of the children over sixteen weeks spent only a reasonable amount of time each day with parents, siblings, or playmates. One to three children at every age level, sixteen to forty weeks, were reported fussy if play with a certain member of the family (usually the father) was not forthcoming at the time of day at which they had come to expect it.

Play relations between siblings appear to be better in the first year than in the second. Until he can walk the new baby cannot

threaten the possessions or playtime of the older child. He remains in one place and the older child can amuse or be amused at his pleasure. Frequently the ability of the younger child to get about changes the situation considerably. Fairly frequent conflicts between siblings were reported in two or three interviews at each age level, fifteen to twenty-four months.

The question as to the place for an infant's play presents some difficulty. From thirty-two to fifty-two weeks the high chair was thought a "good, safe place" for much of the waking time of two or three children at each age level. Certainly this provides far too little opportunity for gross muscular activity. Only in 18.4 per cent of all the interviews, forty weeks to eighteen months, was the use of a playpen or other safely enclosed place recorded. Ideally such a place for play should be of maximum use at these age levels. The floor as a play place was discriminated against in a few instances because the mother feared that it was cold or dirty.

In 17.5 per cent of the interviews with mothers of children fifteen months of age and older it was reported that there were no other children available for playmates. In a very small number of instances this was because neighbors were discriminated against—thought rough or were of a different nationality. In a few instances only older children were available, and the runabouts became very much fatigued in their effort to match the pace set by these older children.

Questions concerning play materials were not presented except by one mother who stated that she found the matter of what toys to buy a real problem. For the most part, few toys were bought, and the women early reported that they found that the children got most pleasure out of simple things which they found about the house, such as pots, and pans, spoons, or sand in the yard.

F. Crying: The evidence accumulated in this study bears out an observation already made by many authors. Children may easily acquire the habit of crying because their mothers interpret this behavior as expressive of acute distress or unhappiness rather than merely communicative of a passing discomfort or desire. We should like to add that the quantity and quality of crying are also dependent upon individual temperamental differences and that the mother of a readily expressive child is, from his birth, confronted with a more difficult training problem (at least during the first year) than the mother of a temperamentally placid infant.

The amount of fussing and crying in the first three months is one of the chief indicators of a child's well-being and general adjustment to life. At four weeks an infant is regarded as well established if he sleeps about eighteen hours out of twenty-four, cries only for brief periods before feedings and perhaps on removal from the tub. In our series only one of six infants intensively studied at four weeks

achieved this ideal. Another who gave every other evidence of satisfactory adjustment, including eighteen hours' sleep, fussed or cried continuously for two and a half hours every evening. Three more cried two to three hours in the early evening and two of these also cried an hour or so in the early afternoon. It was not easy to understand why very young infants whose days are not unlike their nights as far as rest is concerned should characteristically cry in the evening. Does the mother's fatigue at the end of the day play an important rôle?

At six weeks the prolonged evening crying had dropped out for three of the four children just mentioned and was reported in only one of eight infants. It was at eight weeks that the late afternoon crying became frequent. Of seventeen infants, eight cried regularly between 4 and 6 P.M.; after sixteen weeks, one to six children at each age level were reported to fuss or cry habitually in the late afternoon. This was attributed both to fatigue at the end of the longest waking period and to the fact that many infants are conditioned to expect a period of attention from their mothers at the end of the afternoon when the day's work is finished.

That crying serves the purpose of communication between mother and child is brought out by the fact that almost never did the mothers report unexplained crying in children after the period of preliminary adjustment. Only in one interview as early as twenty weeks was it said that a child practically never cried but this was increasingly reported. Fifty per cent of the children at fifty-two weeks were said to cry very little. Very little or no crying was reported in the case of 66 to 85 per cent of the children at each age level after fifteen months. This would seem to have relation to the development of other methods of communication as well as to reflect the fact that in the last analysis crying is not an acceptable form of behavior.

For children from sixteen to fifty-two weeks of age, physical discomfort was held accountable for much of the crying. Discomfort from gas was not referred to after thirty-six weeks. Listing them in descending order of frequency the following physical causes were reported: hunger, fatigue, minor accidents, teething, wet diapers, discomfort due to clothing, heat or cold, illness.

From 25 to 70 per cent of the children sixteen to fifty-two weeks of age cried when they wanted "to be picked up." The test as to whether this was a true interpretation was found in cessation or continuation of crying after the child had been taken up. If crying continued, other causes than mere desire for attention were looked for. Very few other conditions were recognized as causing crying in the first year and these only in one or two instances each. Those mentioned were: thwarted desire to suck the thumb, straining for a toy fallen out of reach, confinement to a play yard, and the presence of strangers.

The explanations as to causes of crying differed markedly in the second year. With the exception of injury, physical discomfort (including hunger and fatigue) was not mentioned. Crying "to get his own way" was reported in 28 per cent of the interviews. The other causes given were: teasing by siblings, removal of a toy, "strangeness," inability to make things do what he wanted them to, discipline, dressing.

The way in which crying was dealt with depended, as noted above, on the interplay of insistence on the part of the child and determination and comprehension on the part of the mother. There was much individual variation in the way in which children cried and quieted. Some began with fussing and became more and more insistent. Others cried sharply from the moment they began. Some shifted instantly from hard crying to complacence while others sobbed and "sulked" for fairly long periods if they were allowed to have a hard cry. [The quality of one child's crying was reported as hysterical, and the mother was convinced that it was uncontrollable.] All the usual methods of quieting were reported, the child was picked up, rocked, quieted by being talked to, with music, or other distraction, by changing the diaper, by nursing or feeding (only in a few instances at a time far removed from the regular feeding time), by giving water. One to three mothers at each age level disregarded crying which they were sure had no good cause.

G. Anger and Fear: Information concerning expressive behavior attributed by the mothers to anger or fear was sought only in the intensively studied infants between sixteen and fifty-two weeks of age. It was here as elsewhere impossible to verify interpretations. Most of the reasons given for anger can be included under two headings: first, physical restraint—thumb or fingers removed from mouth and restrained, restraint imposed by clothing, inability to kick off blankets. Second, thwarting—failure to go to a child or pick him up (by far the largest number of instances reported), failure to provide food or play, removal of toy or food, inability to manipulate an object. In about 18 per cent of the interviews the mothers could recall no occasion on which the child had seemed angry.

Fear was reported (or recognized) very seldom. In 50 per cent of the interviews the mothers could not recall anything which had frightened the child. As might be expected, the larger number of instances up to forty-four weeks were attributed to loud, sudden or unusual noises, such as sudden voices, auto horn, sister screaming, vacuum cleaner, or bells. Second in order of importance were sudden changes in the status quo—persons approaching the infant too quickly, lifting or putting him down too suddenly, or tipping over some article of furniture. The third cause of fear was strangeness in the appearance of people or things (sister in mask, people in glasses, things on wheels).

Fourth, finding themselves in a situation out of which they could not extricate themselves, such as becoming wound up in a blanket; able to pull to standing position but unable to sit down; inability to get off a kiddy car.

H. Thumb-sucking, etc.: Of the twenty children intensively studied only one has emerged as a persistent thumb-sucker. Habits of this nature reported in the other children were: sucking the thumb, fingers, or fist; grinding the teeth; sucking the nipple of the bottle; covering the face with a blanket on going to sleep. A policy of disregard was advised when these habits were reported. In general the mother's attitude was a tolerant one, and the habits were of short duration. Six of the twenty children never had any of these habits even for a brief period. The persistent thumb-sucker mentioned above sucked her thumb most of the time during the first year and, when her hand was not otherwise occupied, was still doing so when seen at twenty-seven months of age. In this case many methods of breaking the habit were tried by the parents who were much irritated by it. It was our impression that this attitude of irritation contributed largely to the continuation of the habit.

CASE REPORTS

(It must be borne in mind that for the purposes of this study the mother's personality or character traits were noted only in so far as they had obvious bearing on the child's behavior. No attempt was made to analyze the sources of her emotional attitudes.)

Example A. A well-handled child.

Baby C. S. was the first child of parents who had married when between twenty and twenty-five years of age.

Mrs. S. was an orderly, conventional young woman. Possessions and their maintenance in excellent condition were important to her. Emotionally she was placid, not expressive, but quietly affectionate. Occasionally playful, never ruffled, she was excellently constituted to care for a child. Interest in the child was well balanced by interest in social life with her husband and meticulous household standards.

Instruction received at the hospital regarding methods of infant care was supplemented by lessons arranged with the visiting nurses. The child was under the charge of a private pediatrician, but the mother wished to avail herself of the psychologic service at the Well Baby Conference.

This mother's ability to organize and her delight in order resulted in a well-planned and executed schedule for the child. C. was temperamentally like her mother and "fell in" with the plans. Mrs. S. understood very well the significance of infant expressive behavior and treated it nonchalantly. The child was never picked up while crying, for instance. She was quieted first with talking or a toy and taken up after she stopped crying. Although this mother enjoyed her baby, development was anticipated with delight, and the child was graduated in good time in each field of behavior. Training for bowel and bladder control was begun early (by the thirty-second week) and the child was regularized by the time she was forty-four weeks old. She was feeding herself at fifteen months of age. Use of a play pen was also begun at thirty-two weeks, and it was used until the child

was about eighteen months of age. Sufficient provision was made for free play in the house and yard so that the adult possessions were not threatened. The need of contemporaries had been recognized, and there was some daily play with other children from about two years of age. She took her place well with such children.

Only one or two minor difficulties were discussed in the course of the interviews. For instance, at about thirty-two weeks the mother noticed that the child was beginning to suck her fingers. She slapped the baby's hand once or twice and the behavior did not recur. Later when the child's fingers were put to her mouth, the mother recognized that her gums were uncomfortable because of teething and was able to ignore it. When last seen at two years and eight months of age, the report was still excellent. The daily schedule had been adapted to the child's growth, and she was very happy in it. The persistent question noted by the psychologist on each of the interview record sheets in the early weeks of the child's life (is the schedule running so smoothly that the changes necessary as the child develops will be difficult to the mother) had therefore been answered in the negative.

Practically no suggestions were made to this mother. She was helped to realize the child's developmental needs at three or four of the age levels and the attitude of tolerance with respect to the finger-sucking was commended.

There must be numbers of other women who are carrying out the training of their children as intelligently as this one. About 50 per cent of the group intensively studied were producing comparably good results, and it was true of about 40 per cent of the group less intensively studied.

Example B. A very immature mother.

Baby A. E. The parents of this child had also married comparatively young. Mrs. E. was plump, jolly, and soft-hearted. Her pleasant dimpled smile had been reproduced in her baby. Although good-natured for the most part, she did not lack temper. She was guided by her feelings rather than by her intelligence. She was influenced by various superstitions. She had a naïve belief in the reappearance of her own or her husband's acquired characteristics in the child (such as a dislike for asparagus).

The baby was born in a small private hospital, and the mother was given a little training in his care there. She had previously learned something about babies by helping her sister with the care of hers. The visiting nurses had supplemented this information. Of the extraverted type, she knew from much chattering the methods of care of many of the mothers in her immediate neighborhood.

A. was consistently well developed mentally. He was fundamentally good tempered but easily discovered the "soft heart" of his mother and occasionally made adaptive use of expressive behavior. She thought him sensitive when disciplined, rediscovering herself in this, saying, "No one was ever able to say 'boo' to me." She was more capable of dealing with him as a little infant than she was as he grew older.

Difficulties were encountered in practically every field of behavior, though in many instances a question raised at one interview had been forgotten by the next. In the feeding situation solids met with resistance from the first. Cereals were regurgitated when first presented between sixteen and twenty weeks. By twenty-four weeks cereals had been accepted, but vegetables were meeting with the same resistance. Each time the mother was advised to "continue to offer the rejected foods calmly and persistently." At twenty-one months the use of a bottle was

still permitted, because the child protested so vehemently if it were removed. Later she said, "Ever since he gave up his bottle on Ash Wednesday for Lent, he has had a blanket to go to sleep with."

Regularity of sleeping hours was not insisted upon during the day when the child was a very young infant. He slept when he felt like it and other necessary things were done when he happened to be awake. None of the suggestions made changed this situation, but by the time he was twenty-eight weeks old the mother found for herself that regularity made her housework easier and was arranging naps accordingly.

Training with respect to bowel and bladder control was not begun until A. was forty-four weeks of age and was then carried on in half-hearted fashion. First it was put off until the child could sit alone, then until the proper bathroom equipment could be obtained.

The child was always overprovided with toys. He early succeeded in spending his waking hours in the room with his mother. At three years his play interests were of unusually short duration.

When the child was three years old, methods of discipline remained impulsive, opportunistic and emotional. The child made special effort to finish a meal when told a horse with whom he wanted to be friendly would be more interested in him if he did so, and was distinctly disappointed when the horse was as bored as ever.

The further development of this child will be watched with interest. Although his mother is genuinely fond of him, she is so immature that one feels a child has been set to raise a child. Granted a strong will and sufficient independence, a little more development may see A. taking the helm long before his judgment is sufficiently well developed.

This example represents about 11 per cent of the children studied.

Example C. An overanxious mother.

Baby E. J. E. J. and her mother were both tense, readily expressive individuals, and clearly the difficulties arose in part from the interplay of these two like personalities.

Mrs. J. was a sensitive, intelligent woman. There was an older child in the family, but E. was her father's favorite. He was often reported as responsible for this or that care method, usually those involving very protective measures, as he feared for her physical safety. Mrs. J. was constantly overtired, constantly puzzled and worried by the child's behavior. She was interested in the periodic observations, but also welcomed the two- to three-hour interval as a physical rest. She felt safe about the baby and herself relaxed at this time as she rarely did at others, though at the clinic she feared situations which were accepted calmly by the other mothers, such as leaving the child alone in the play pen or crib, lest she get arms or legs entangled in the bars.

E. was an exceedingly alert, intelligent child. She was characterized as superior when first seen at twenty-eight weeks, and this diagnosis was borne out by all subsequent observations. She was last seen at two and a half years of age. She was constantly active and entirely fearless—traits which formed the basis of her father's fears when she reached the climbing stage. She was physically attractive and always interesting in her responsiveness, so that the devotion she commanded from her family was readily comprehensible.

E. was one of the radicals who from the first preferred solids to milk. The mother was perpetually worried because the child took less than the prescribed quantity of milk. She therefore fell into the habit of offering it to her at odd times of the day in the hope that more could be "got in." The child gained

consistently at a slow rate and was always thin and wiry. The mother's worry was increased by a statement made by her pediatrician that any child could be fattened, because it seemed impossible to realize this end for either of the children in the family. Time showed that the mother's report about the amount of food consumed by the child varied from month to month. It finally seemed to be indicated that whether the report was optimistic or pessimistic depended somewhat on the mother's own state. The last report (at two and a half years of age, given as if it were a discovery by the mother, though the point had been stressed by both pediatrician and psychologist) was that the child ate better when she was prevented from eating between meals.

Although E. slept fairly well at night, the brevity of the daytime nap was a further source of worry. Because of the child's hyperactivity the mother was afraid to leave her alone until she was actually asleep. At night she was in the parents' room and could be protected as soon as she woke. At nap time the mother felt the need of hovering near the child's carriage lest she wake and have a fall. She seldom slept more than half an hour at a time in the early weeks, a little longer at one year of age. Once, when the mother was persuaded to leave the child alone at the clinic, she slept for an hour and a half, suggesting that the constant presence of people was a contributing factor to the brevity of her naps. At two and a half years of age, she was taking no daytime naps and was said to be a restless sleeper at night.

Training for control of bowel and bladder was not begun until after the child was thirty-two weeks of age. The mother was waiting to secure perfect equipment. Regularizing of bowel movement was accomplished easily, and complete regularization of both functions was well under way at one year of age.

The bathing and dressing period was again one of stress for the mother because the child was hyperactive. During the rest of the day the mother was constantly afraid to have E. out of her sight, as she was fearless about climbing and was not inhibited by falls. Wherever the mother was working, there she felt that she must have the child. Because naps did not provide any period of peace, the wear and tear were constant. A padded play pen provided some relief until E. acquired the ability to climb out of it shortly after she was a year old. At two and a half years of age play still presented the chief problem. E. fought with all the children in the neighborhood. She hit and bit to get what she wanted and was feared by the other mothers though she was smaller than most of the children. She was usually allowed out only when her sister could be there. She was not afraid to approach any animal. Taken to bathe in the Sound, she repeatedly went out beyond her depth when left to herself.

Though the child was for the most part cheerful and socially agreeable, she possessed a potent weapon in crying. Not only did the mother feel that crying must be given in to lest an elderly neighbor who shared the house be disturbed, but also it was felt that there was an hysterical quality about the crying. On one or two occasions when she was allowed to cry, quieting was a very difficult process and was not accomplished even by giving the child the thing for which she had originally cried. At two years of age she fell and fractured some part of her shoulder. For two days she said she was a "broken girl" and that she "could not walk." At the end of that period her sister mentioned something interesting at the window; E. forgot her disability and ran across the room to investigate. The family pediatrician considered this a true hysteria.

Here, then, are many seeds of difficulties which may bear fruit. Many people have talked sensibly to Mrs. J. but her ability to control herself is constantly threatened by the factors outlined above. Pos-

sibly when the child's very active mind is busied at school several hours of the day and the mother is insured a few more peaceful hours, the situation may improve. One of the necessities of the future would certainly appear to be the provision of holidays for such mothers from such children.

Fourteen per cent of the children studied represented somewhat similar situations.

SUMMARY

1. Fifty-nine children were studied at a Well Baby Conference organized by the Visiting Nurse Association in Hamden, Connecticut, with a desire to determine, first, methods of child care actually in operation; second, to define problems in management encountered by the mothers; and third, to arrive at conclusions with respect to the function fulfilled by a psychologist in such a conference. The group studied was a little above the average with respect to intelligence and economic level.

2. On the whole, methods of child care were encouragingly good, indicating that education in this field is making headway. It was increasingly apparent, however, that individual differences in infants (with respect, for instance, to food and sleep requirements) are too little stressed in the literature and in common parlance, with resultant undue anxiety on the part of those mothers whose infants deviate most from the accepted standards, even though such deviations fall within normal limits of variation. Reassurance was often necessary to the effect that even marked deviations could be considered as characteristic of a given child and did not constitute a threat to his general well-being.

3. A very few instances of most of the problems of management recorded in the literature were encountered in the course of the study. Of the forty-six children studied more than once, twenty-seven presented practically no problems, thirteen presented a succession of transient minor problems, six evidenced persistent difficulties. The prognosis with respect to ultimate adjustment was thought to be good for thirty-three of these forty-six children, questionable for nine, and poor for only two of them.

4. The existence of a preliminary period of difficult adjustment in the first two or three months of life has also been insufficiently emphasized in the literature. That the child needs time to adjust to rhythmic conditions of living and the mother, in her weak and emotional state, to her new responsibilities was repeatedly evidenced in the course of this study. The importance of sympathetic supervision and advice during this period cannot be overemphasized.

5. The situation with respect to the function of a psychologist in a Well Baby Conference defined itself as follows:

a. The way in which appointments were kept indicated that the mothers were very much interested in the information which the psychologist had to give them with respect to the infant's increment of mental growth.

b. Many of the suggestions made by the psychologist failed to be incorporated in the mother's program of child care until her own practical experience emphasized the need. The fact that suggestions respecting physical care were eagerly sought for and immediately put into practice, while suggestions regarding care from the psychologic standpoint were seldom sought, indicates that education in this field lags far behind that in the physical field. Further community education with respect to the type of question a psychologist is equipped to answer is necessary before such an individual in such a situation can be of maximum usefulness.

6. In the last analysis the mother's attitudes in child care situations are of first importance in determining the child's behavior. Not only must she be intellectually receptive to current concepts of child care, but also she must be so constituted with respect to temperament and so adjusted in her own emotional life that she can put her intellectual concepts into practice if she is to be successful in the management of her child.

INFANT MORTALITY: CAUSES OF DEATH DURING THE FIRST YEAR OF LIFE AS DETERMINED BY NECROPSY IN 517 CASES

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IN A REVIEW of the protocols of postmortem examinations of infants and children during a period of three and a half years (July, 1928, to January, 1932) in the Department of Pathology at the University of Minnesota, I noted that approximately 58 per cent of the examinations were of infants who died in the first year of life.

The subjects examined were from various hospitals in Minneapolis and St. Paul, and in many instances they were referred by physicians in private practice in these cities. The selection, therefore, is a fair index of the infant mortality in these cities during this period.

A classification was made according to the primary cause of death in certain age groups arbitrarily chosen (Table I). No attempt was made to describe in detail pathologic changes in any single case or group of cases, but rather to list as accurately as possible the primary cause of death in each case. Because of the frequency of complications the primary cause of death was sometimes perplexing, and in such cases the sequence of events in the illness as obtained from the hospital records was utilized. Infant mortality is a comprehensive term, and included in this series are all cases in which the infant breathed after delivery even though the respirations were feeble. Infants born with hearts which were beating but who never breathed were considered to be stillborn. If the infants died within the first seven days after birth the factors associated with pregnancy and labor undoubtedly were most significant, but a consideration of these has been omitted purposely. Of those who died in the first twenty-four hours after birth, twenty-one died before the end of the first hour; nine as a result of trauma at birth, nine of premature birth, one of fetal ascites, one of congenital syphilis and one, prematurely born, of localized meningoencephalitis. In this group twelve twin infants were examined. In three cases both twins died, in six cases one twin survived and in one case one twin was stillborn.

Trauma at birth was the cause of death in 102 cases (19.7 per cent) which is 30 per cent of the deaths during the so-called neonatal period, the first four weeks of life. Deaths due to injury at birth were divided into subgroups, depending on whether the infant was born prematurely

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TABLE I

CAUSES OF DEATH DURING THE FIRST YEAR OF LIFE IN 517 CASES, AS DETERMINED BY NECROPSY

	FIRST DAY			DAYS, 2 TO 7			7 DAYS TO 1 MONTH			MONTHS, 1 TO 6			MONTHS, 6 TO 12		
	BOYS	GIRLS	TOTAL	BOYS	GIRLS	TOTAL	BOYS	GIRLS	TOTAL	BOYS	GIRLS	TOTAL	BOYS	GIRLS	TOTAL
Trauma at birth (full term)	24	18	42	9	6	15	2	1	3						
Trauma at birth (premature)	22	11	33	3	6	9									
Premature birth	46	30	76	7	5	12	3	5	8		1	1			
Postmature birth	1	2	3												
Congenital anomalies	4	4	8	7	6	13	3	3	6	8	8	16	1	2	3
Pneumonia	6	2	8	7	6	13	17	7	24	27	13	40	13	7	20
Congenital syphilis	1	1	2				1		1	3	1	4		1	1
General fetal dropsy		3	3												
Asphyxia	3		3		1	1	1		1		1	1	1		1
Pulmonary edema	1	1	2												
Peritonitis				3		3	1		1	1	1	2			
Duodenal ulcer, melena				2	1	3									
Jaundice				1		1									
Mastoiditis					1	1	2		2	6	5	11	9	2	11
Septicemia								3	3	2		2	2	1	3
Marasmus							4	3	7	3	1	4		1	1
Erysipelas								2	2	1	2	3			
Omphalitis							2	2	4						
Impetigo contagioso								1	1						
Subacute bacterial endocarditis							1		1						

TABLE I—CONT'D

	FIRST DAY			DAYS, 2 TO 7			7 DAYS TO 1 MONTH			MONTHS, 1 TO 6			MONTHS, 6 TO 12		
	BOYS	GIRLS	TOTAL	BOYS	GIRLS	TOTAL	BOYS	GIRLS	TOTAL	BOYS	GIRLS	TOTAL	BOYS	GIRLS	TOTAL
Cardiospasm							1		1						
Sinusitis								1	1						
Pyelonephritis							1		1				2	3	5
Tuberculous meningitis										1		1		1	1
Tuberculous peritonitis													1		1
Generalized miliary tuberculosis										1	2	3	1	1	2
Meningitis, epidemic													1		1
Meningitis, nonepidemic										3	2	5			
Intussusception										1		1	5		5
Diphtheria										2		2		1	1
Enterocolitis										1	1	2	1	1	2
Pyloric stenosis										3	1	4			
Sarcoma										1		1	1		1
Intestinal obstruction											2	2			
Portal cirrhosis													1		1
Mesenteric thrombosis													1		1
Glomerulonephritis													1		1
Progressive muscular atrophy													1		1
Undetermined	5	1	6	3	1	4		2	2	6	9	15	4		4
Total	113	73	186	42	33	75	39	30	69	70	50	120	46	21	67

or at term. This distinction is important. Chase noted that if birth occurs prematurely, splits in the tentorium are relatively more common than if birth occurs at full term. This, he has shown, is due to immaturity of the fibers of the dural septums, the delicacy of the blood vessels and the predominance of fibroblasts in the tentorium cerebelli and falx cerebri at this period. Maturity and delivery past term are factors necessitating operative procedures in which trauma is not uncommon. The maturity or prematurity of these infants was calculated in accordance with Hasse's Law from the crown-heel length in centimeters. In all cases intracranial injury, laceration of the falx cerebri, tentorium cerebelli, or both, with or without hemorrhage were demonstrated. In four cases there was additional trauma; in one case subcapsular hemorrhages of the liver and bilateral hematoma of the suprarenal glands, in another bleeding into the neck, myocardium, pleura and suprarenal glands, in one hemoperitoneum in which the source of the bleeding was not found, and in another bleeding in and about the umbilicus.

In forty-eight cases, in which data from the obstetric records were available forceps had been used in nineteen cases, in twenty-four cases delivery was spontaneous and in five, version and breech extraction were done. In seven cases developmental anomalies occurred, four were of the urinary tract, two were hydrocephalus and one was esophageal stenosis and polydactylia. Adair, in a series of 172 neonatal deaths found that 55 per cent of the premature infants and 56 per cent of the mature infants died from injury at birth. In Cruickshank's larger series of cases gross intracranial hemorrhage was noted in 20 per cent; Lyon and Bemis found intracranial injury the cause of 28 per cent of neonatal deaths.

Premature birth was the cause of death in ninety-seven cases (18.7 per cent). Except for the immaturity, as calculated from the length of the body, no obvious primary cause was found. It was in this group particularly that certain maternal factors were significant. Most of the premature infants died in the first twenty-four hours. In three cases malformations were present; two were of the genitourinary apparatus, and one was a harelip and cleft palate.

In three cases, the length and weight of the infants and the calculated duration of pregnancy indicated delivery past term as the cause of death.

The cause of death in forty-six cases (8.8 per cent) was congenital malformation. In twenty additional cases (3.8 per cent) in which death occurred from another primary cause, congenital anomalies were found. Congenital anomalies in this entire series comprised 12 per cent. Epstein found defects in development to be the cause of death in 25 per cent of cases in the first month, and in 9 per cent of cases in which death occurred after the first month and before the end of the first

year. In the cases in which anomalies were present but in which there was some other primary cause of death, eight of the malformations were of the genitourinary apparatus, three were hydrocephalus, three were congenital defects in the heart, two were harelips and cleft palates, and one was porencephalia. Two infants were mongolian idiots. One infant was a pseudohermaphrodite, one had club feet and one polydactylism. In most of the cases multiple defects in development were present.

Twenty-three of the infants were boys and twenty-three were girls. The most frequent malformation was hydrocephalus. Of the thirteen cases of hydrocephalus ten were of the obstructive type. Three had been treated surgically just prior to death. In two cases empyema of both ventricles and meningitis occurred; in two there was terminal pneumonia. There were three cases of the communicating type of hydrocephalus. Porencephalia occurred in two cases and was the only finding of note. One anencephalic monster lived three hours after birth.

Of the congenital defects of the heart the most common was a patent interventricular septum. Malformations of the genitourinary tract are probably relatively more common than the incidence in these forty-six cases would indicate. Four infants, between the ages of one month and six months, had complete obstruction of the common bile ducts. One infant had atresia of the ileum and three of the duodenum. Enterostomy had been performed shortly after birth on one of these infants. The immediate cause of death was peritonitis. The bowel of one infant had ruptured spontaneously, with resultant peritonitis. Two of the infants with atresia of the duodenum had acute suppurative pancreatitis. The infant with imperforate anus had been treated surgically, and during this procedure the sigmoid perforated and peritonitis caused death. The infant with ectopic viscera had acute peritonitis and pericarditis; death occurred on the seventh day. When defects of the diaphragm had occurred, abdominal viscera herniated into the thoracic cavity (Table II).

Pneumonia was the cause of death in the largest group (20 per cent) of the cases. The term acute pneumonia has been employed in the classification rather than the usual distinction of lobar pneumonia and bronchopneumonia. Because of the frequency of complications these cases were the most difficult to classify. As a general rule the deaths listed under pneumonia include those in which pneumonitis clinically preceded the conditions which otherwise would be primary causes of death. Involvement of the pleura was not noted in any of the cases in which death occurred within the first twenty-four hours, and it was noted in only one case in which death occurred within the first week. In seven cases empyema was a complication, in three peritonitis, in one case meningitis, in one suppurative pericarditis and in one enterocolitis. In three cases intracranial injury was sustained at birth, and

TABLE II

FORTY-SIX CASES IN WHICH MALFORMATION WAS THE PRIMARY CAUSE OF DEATH

	AGE AT TIME OF DEATH				
	FIRST DAY	2 TO 7 DAYS	7 DAYS TO 1 MONTH	1 TO 6 MONTHS	6 TO 12 MONTHS
Heart and Circulation					
Deformities aortic valves		1			
Single auricle and single ventricle		1			
Patent intraventricular septum		3	3	2	2
Transposition of great vessels			1		
Dextrocardia				1	
Coaretation of aorta				1	
Single auricle				1	
Pulmonary stenosis			1		
Nervous System					
Hydrocephalus, obstructing	2	1	1	4	2
Hydrocephalus, communicating		1	1	1	
Spina bifida	2	1		2	1
Meningo-encephalocele	2				
Porencephaly				2	
Anencephaly	1				
Hypoplasia cerebellum		1			
Urinary System					
Hydronephrosis and hydro-ureter	2	1	3		
Polycystic kidneys	1				
Congenital absence of kidney		1	1		
Hypospadias of kidney	1				
Hypospadias	1				
Imperforate vagina	1				
Gastrointestinal Tract					
Atresia band stenosis of the bowel		1	3		
Imperforate anus	1	1			
Stenosis of the common bile duct				4	
Miscellaneous					
Bicornuate uterus		1			
Polydactylism		2			
Defective diaphragm	1	2			
Cleft palate; harelip	2	1			
Club feet	1	1	1		
Achondroplasia		1			
Ectopic viscera		1			
Pseudohermaphrodism	1				
Anomalous lobulation of lung		1			

in one case there was thrombosis of the right renal vein. Nine infants were born prematurely, and one infant had pneumonia before delivery. Eight infants had pus in one or both mastoid processes, and four had simple otitis media. McNeil found the highest death rate from pneumonia occurred during the first year of life, although in most of his cases it occurred in the period from one to two years.

Eight infants died from congenital syphilis. The criteria for diagnosis were one or more of the following: demonstration of the spirochete in the tissues, a positive serologic reaction, or syphilitic osteochondritis. One infant had the characteristic pneumonia alba.

There were three cases of generalized fetal dropsy. Death of all infants took place within the first twenty-four hours. Causes for this abnormality have been listed as syphilis, defects in the circulatory system, cardiac failure, absence of the ductus venosus, pressure from abdominal tumors or distended urinary bladders and hypoplasia of the urinary apparatus. No apparent cause was found in this series of cases.

Seven deaths were a result of asphyxia. In two instances the head had remained on the perineal floor for fifteen minutes or more following a breech delivery, and in one instance deep obstetric anesthesia had been used. One infant aspirated a large amount of milk and one aspirated a foreign body which produced severe tracheobronchitis. In two cases no cause for the asphyxia was found.

Two infants who died in the first twenty-four hours had severe pulmonary edema. Perhaps these two deaths might have been grouped as indeterminate.

Peritonitis was the primary cause of death of six infants. One infant was two days old, who had a strangulated inguinal hernia, with gangrene and perforation of the bowel. One infant had an abscess of the breast. One died following an end-to-end anastomosis of the small bowel for intestinal obstruction of undetermined etiology. Two infants died from primary peritonitis.

Three infants had one or more ulcerative lesions of the duodenum with severe hemorrhage; all of these were aged less than one week.

One infant died from severe icterus presumably, since this was the only significant postmortem finding. Details of the clinical history were not available.

Mastoiditis with various complications caused the death of twenty-five infants. Two had associated staphylococcic septicemia, three had streptococcic peritonitis, one had lateral sinus thrombosis; four had meningitis: one pneumococcic, one streptococcic and one influenzal; the organism in the fourth case was not identified. Fourteen of the infants had pneumonia. Cultures of the pus from the mastoid processes were not made as a routine. Lierle and Potter, in a series of 200 cases of mastoiditis of infants found a mortality of 34 per cent. The mortality

in these cases in which operation was performed was 48 per cent. The most common causes of death following operation were bronchopneumonia, collapse, septicemia and thrombosis of the lateral sinuses.

Eight deaths were due to septicemia or pyemia. The staphylococcus was isolated in four cases of multiple abscesses of the lungs, kidneys and other organs. In one case the condition followed adenoidectomy and irrigation of sinuses, and death was due to the streptococcus. In three cases the organism was not recorded.

Marasmus was the cause of death in twelve cases. Five of the infants were born prematurely and one had spastic diplegia. Five deaths resulted from erysipelas and complications. Two infants had streptococcic peritonitis and two had pneumonia. Four deaths resulted from omphalitis with complications. One of these infants had multiple abscesses in the liver, and two had generalized peritonitis; another had an old intracranial injury. The cause of death of three infants was impetigo contagioso, subacute bacterial endocarditis, and cardiospasm. The last infant had vomited since birth and the distal part of the esophagus was markedly dilated. One infant died from purulent sinusitis complicated by a retrobulbar abscess and meningitis.

Six infants died from pyelonephritis. Only one infant was aged less than six months. One infant had thrombosis of both renal veins and two had hydronephrosis and hydroureter with infection of the entire urinary tract.

Two infants had tuberculous meningitis, one had tuberculous peritonitis, and five had generalized miliary tuberculosis. Four infants apparently had primary, nonepidemic meningitis, in two instances from streptococcus and in one instance from pneumococcus. The organism was not isolated in the fourth case. One infant died from epidemic meningococcic meningitis.

Six infants died from intussusception; all were girls. The intussusception had been reduced surgically in five instances; four infants had peritonitis and one had hemoperitoneum. Laryngeal diphtheria caused three deaths. Enterocolitis caused four deaths. In one instance Shiga's bacillus was isolated; the other organisms were nonspecific. Pyloric stenosis was the cause of deaths of four infants on whom the Rammstedt operation had been performed. One infant died from peritonitis and the others from bronchopneumonia.

Two infants died from malignant lesions. One of the infants was a boy, aged eight months. Tumorous tissue was found in the lungs, liver and pancreas; it was highly cellular and infiltrating and was composed chiefly of round cells. There was no tendency to form structures resembling normal tissue. The growth may have been primary in the pancreas or liver, but the extensive process in the liver made this the most likely primary site. The other infant was a boy, aged five months.

The tumor was a round-cell sarcoma, primary in the suprarenal glands, with metastasis to the skull and forearm.

Intestinal obstruction caused two deaths. In one instance there was rotation of the colon and small intestine about the root of the mesentery; in the other extensive duodenal bands had formed.

In single instances deaths were due to hepatic cirrhosis, mesenteric thrombosis, progressive muscular atrophy and subacute glomerular nephritis. Splenomegaly was associated with cirrhosis of the liver; the Wassermann reaction of the blood was negative.

The cause of death of thirty-one infants was undetermined. Approximately 50 per cent of these were aged from one month to six months. The postmortem and clinical data in all of these cases were essentially negative.

COMMENT

The observation of this series of cases has made apparent that further progress in the reduction of the lamentably high mortality among infants depends on more exact knowledge of the causes and factors most concerned. This report is not offered for the purpose of forming fixed conclusions as to the causes of such mortality in all localities. It does, however, offer an index to the relative frequency of the various causes in a rather extensive northwest metropolitan area during a period of three and a half years.

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THE METABOLISM OF IRON IN THE ANEMIA OF PREMATURE INFANTS

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IN INFANTS born prematurely, the erythrocyte count and hemoglobin fall to a low level and return to normal during the first six months of life.

Finkelstein[1] states that prematurely born infants develop anemia in spite of treatment and recover in due course of time without special therapeutic measures other than the treatment required because of their immaturity. In this clinic[2] it has been shown that in general an anemia develops in premature infants up to about ten weeks to twelve weeks of age, after which the blood tends to return to normal, spontaneously. It has been noted also that the amount of reduction in red blood cells and hemoglobin varies inversely as the length or weight of the infant at birth, or, in other words, directly with the degree of prematurity. The red cells and hemoglobin have a coincident fall, and the color index remains close to unity.

Many investigators believe that premature infants are lacking in the mineral elements. Mineral balance studies[3, 4, 5] made on pregnant mothers have shown that during the last three months of intrauterine life a large proportion of the various inorganic salts is retained, and it is assumed that these are all utilized by the fetus. In contrast to this, analyses of fetuses[6, 7] have shown that throughout pregnancy there is the same relative amount of the various inorganic elements as are found in the fully mature fetus. The larger positive balance in the last three months of pregnancy is thought to be due to the rapid gain in fetal weight during that period. Quantitative analyses for iron have shown that fetuses and newborn infants contain approximately three times as much of this metal in proportion to body weight as does the child of one year or the adult.[8] In other words, there is presumably enough iron reserve in the newborn infant, whether born prematurely or at term, to allow for a tripling of the birth weight without the requirement of additional iron from the food, providing there has not been a loss by excretion or hemorrhage.

The development of anemia in premature infants with subsequent spontaneous recovery has not been adequately explained, although

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various theories have been advanced. In order to secure further information concerning the phenomenon of the development of anemia in premature infants, iron balance studies were carried out for twelve periods, of three to six days each, on seven premature infants of varying ages. The pigment excretion of these infants was recorded throughout their stay in the hospital.

METHODS

The infants were placed on metabolism beds designed by Dr. Gamble. The urine and stools were collected in porcelain containers. The urine and stool specimens from male infants were collected in separate containers. At first the attempt was made to keep the specimens from females separate, but this was found to be impossible in small infants. Granite ware containers were not used because chipping of the container with consequent rust formation might produce considerable error. Spatulae and all crucible and evaporating dishes were of porcelain. The only metallic articles used in any of the processes were platinum crucibles for the ashing. Carmine was given to mark the stools at the beginnings and end of the observation period. Each patient was kept on a constant twenty-four-hour diet throughout the experimental period. Aliquot samples of the diet, breast milk or formulas, were analyzed and the intake was computed in this manner.

The amount of iron was determined by the amyl alcohol method described by Josephs,[9] which is a modification of that given by Marriott and Wolf.[10] This method was found to give results agreeing within 2 per cent on theoretical solutions as well as yielding good duplicate values on unknowns.

The method of following the pigment excretion is described in another communication[11] and need not be given here.

RESULTS

The data on iron balance and blood findings are recorded in Table I. Nine of the twelve determinations show a positive iron balance and three, a very small negative balance. The average balance per day is +0.10 mg. Fe.

The three determinations giving a negative iron balance show a difference of intake and output of such slight degree that for all practical purposes they may be taken as representing equilibrium.

Patient 6, weighing 4-5 pounds, had a very high positive balance. The breast milk had an exceptionally high iron content, and the infant was evidently utilizing most of it.

All the infants at the time of the test periods showed some anemia as evidenced by a decrease of both the red blood cells and the hemoglobin. The color indices were high and approached unity. The anemia was more pronounced in the infants who were smaller at birth and increased with age in these patients as long as they were followed in this series.

The data on pigment excretion are found in Table II. The stool urobilin and bilirubin excretion was not excessive as compared with my former studies.[11] As in the former investigation, the pigment ex-

TABLE I

EXCRETION OF IRON

PATIENT	BIRTH WEIGHT POUNDS	DATA PERTINENT TO BALANCE STUDY PERIODS						IRON METABOLISM AS FE DURING PERIOD					
		AGE AT BEGINNING WEEKS	LENGTH IN DAYS	HB. PER CENT* AT BEGINNING	R.B.C.* AT BEGINNING	COLOR INDEX	TYPE OF FEEDING†	STOOL MG. FE	URINE MG. FE	TOTAL EXCRETION MG.	INTAKE MG. FE	BALANCE PER PERIOD MG. FE	BALANCE PER DAY MG. FE
1	2 5/16	7	6	53	2.7	0.98	Br.	1.131	0.2105	1.3415	1.92	+.5785	+.0964
		9	5	52	2.8	0.93	Br. + F.	1.024	0.226	1.250	1.7045	+.4545	+.0909
2	2 14/16	3	6	64	3.5	0.91	Br.			1.545	1.509	−.036	−.006
		7½	6	57	3.0	0.95	Br.			1.295	1.843	+.549	+.091
		10	5	58	3.0	0.97	Br.			1.426	1.738	+.312	+.062
3	3 10/16	13	6	60	2.3	1.3	F.	2.682	0.1644	2.8464	2.7891	−.0571	−.0085
4	4	3	6	74	3.9	0.95	Br.			1.376	1.95	+.583	+.097
		5	6	66	2.6	1.3	Br.			1.54	2.15	+.560	+.093
5	4 2/16	2	6	68	4.4	0.77	Br.			1.57	1.605	+.035	+.006
		4	5	69	4.0	0.86	Br.			1.482	1.470	−.012	−.0024
6	3 14/16	4½	3	80	4.6	0.87	Br. + F.			0.94	2.54	+1.60	+.53
7	3	11	3	50	2.8	0.89	F.			1.23	1.74	+.51	+.17
													Average +.10

*Hemoglobin Newcomber method; R.B.C. in millions.
†Br. means breast milk; F. means formula.

TABLE II

DAILY AVERAGE FOR FIVE-DAY PERIODS OF PIGMENT EXCRETION IN STOOLS, EXPRESSED AS MG. UROBILIN PER DAY

CASE	PERIODS									
	I	II	III	IV	V	VI	VII	VIII	IX	X
1	I*	I*	1.3	1.1	1.2	0.3	0.4	0.3		
2	I*	I*	Trace	I*	0.9	0.5	0.6	2.1	1.2	
3	1.8	3.8	3.2	0.5	0.6	0.7	2.8	0.6	1.4	1.0
†4	6.6	1.2	1.0							
5	3.3	1.5	6.0	2.7						
8	0.3	0.5	0.4	0.3	0.2	4.0	4.6	2.2	2.2	1.6
9	0.3	0.3	0.3							
10	0.6	0.4								

*The value I represents a small excretion of bilirubin.
†High excretion due to collections starting at seventh or eighth day of life.

creted in the stools of these infants during the first few weeks of life was urobilin in some and bilirubin in others. After the infants reached two months of age the only pigment found on extraction of the stools was urobilin.

DISCUSSION

These results differ from those of Lichtenstein[12] who carried out iron balance studies on prematurely born infants. He found that there was a negative iron balance unless excessive amounts of iron were fed. Our methods of analysis are different but coincide in giving approximately the same values for iron in breast milk. As the prematurely born infant develops anemia in spite of treatment, and recovers spontaneously, it does not seem reasonable that a negative iron balance could occur for any considerable period.

The data on pigment excretion do not indicate excessive hemolysis and, though Whipple[16] states that urobilin excretion is not quantitatively related to hemoglobin destruction, still there is a definite proportional relation between the two in conditions with excessive blood destruction such as hemolytic jaundice and sickle cell anemia.

The course of the anemia, the iron retention, and the low stool pigment excretion in premature infants indicate that the condition is not the result of blood destruction.

Inadequacy of the milk feeding as a source of iron has been considered an important factor. Experimentally a milk diet has been shown to be effective in producing anemia in rats.[13, 14] In rats, however, at the age which permits the use of an experimental diet, the reserve supply of iron is not so high as in infants. It has been shown in the newborn animals that the iron reserves are directly proportional to the usual duration of the suckling period,[8, 15] and consequently animals maintained on a milk diet beyond the suckling period will in time become anemic. This phenomenon is seen in the human being as well as in the experimental animal.

Anemia due to low iron intake, such as is seen in a wholly milk diet, has certain characteristics. It is present until the diet is corrected by the addition of iron or iron-containing foods. It also shows a distinctive blood picture. The red cells are only slightly affected in regard to numbers, but the hemoglobin is markedly reduced, and as a consequence there is a hypochromic anemia with a low color index.

In contrast to this picture, the anemia of prematurity presents equal diminution of red blood cells and hemoglobin with a color index approaching unity. This form of anemia also develops in spite of iron therapy and recovers spontaneously. From these two facts it appears probable that anemia of prematurity is not a result of inadequate iron supply in the milk.

The positive iron balances recorded are not high, and the average figure of +0.10 mg. per day equals 30 mg. of hemoglobin (100 gm. of hemoglobin contain 0.34 gm. of iron).[18] This would not be sufficient to maintain new hemoglobin formation to make up for the increased blood volume coincident with growth.[17] However, the high iron content of fetuses and newborn infants should[6, 7, 8] provide adequate reserve to meet this requirement.

The findings enumerated above support the following points: (1) the iron balance tends to be positive or at equilibrium; (2) the pigment excretion is not excessive; (3) the anemia presumably is not hemolytic in origin; (4) it is apparently not dietary in type; (5) there are available iron reserves; (6) recovery is spontaneous.

Assuming that the anemia of prematurity is not due to some of the more usual causes of anemia, indulgence in speculation concerning its cause may be permissible. There are at least two possibilities immediately presenting:

(1) The anemia may be a direct outcome of the physiologic immaturity of the bone marrow. This is an all inclusive hypothesis and cannot be confirmed or disproved with our present knowledge.

(2) The second possible factor in the production of the condition is a more specific branch of the first. As has been shown above, the anemia is not due to the want of available building stones for hemoglobin production but, rather, to failure of their utilization. It may be that this failure of utilization occurs because there is insufficient bone marrow to produce enough red blood cells. With growth this incapacity is overcome.

That this may be the case is suggested by the anatomic development of hematopoiesis in the fetus. Blood formation begins in the liver and spleen in early fetal life. For a time in the fetus, these organs are the only source of blood formation, but later, as the bones form from the original cartileges, medullary space is produced. As this medullary space enlarges, the production of blood cells increases in this site and decreases in the liver; so normally in the full-term infant hemato-

poiesis has practically all been taken over by the bone marrow. Analyses of newborn infants have shown that the bone marrow is approximately 2.3 per cent of the body weight.[19]

The prematurely born infant requires extramedullary hematopoiesis to supply an adequate quantity of blood cells, and extramedullary hematopoiesis may be demonstrated in infants delivered at term. It is pronounced in prematurely born infants and is directly proportional to the degree of prematurity, being much greater in the smaller infants.

In a premature infant that survives, the ductus venosus closes and the liver becomes the route of the portal blood. The liver discontinues the function of hematopoiesis and assumes the usual extrauterine functions. Thus the premature infant is deprived of this source of hematopoiesis which would otherwise, as in utero, sufficiently augment the bone marrow so that blood cell production would be adequate.

It also seems likely that the actual amount of available bone marrow space is an important factor in the development of anemia in prematurely born infants. In adults, blood formation, sufficient to maintain a constant level under normal conditions, is confined chiefly to the flat bones and the metaphyses of the long bones. The diaphyses of the long bones contain fatty, inactive marrow which is capable of active blood formation in response to various stimuli. On the other hand, in infants all of the available bone marrow space is utilized to maintain the normal levels. The bones of a premature infant are necessarily smaller than those of a normal full-term infant and have proportionately smaller bone marrow cavities. As the infant grows these spaces enlarge; and, if this factor of available space had any influence on the adequate production of blood cells, the anemia should recover spontaneously when the infant's size increases, with the concomitant increase in the bone marrow space.

That the lack of bone marrow space may conceivably be one of the factors in the production of the anemia of prematurity is an attractive hypothesis, although unsupported by any direct observations. If this hypothesis is correct, it offers an explanation for the relation of the anemia to the degree of prematurity, for the type of the anemia and for the spontaneous recovery.

CONCLUSIONS

The results of studies of iron balance and pigment excretion in a series of premature infants tend to establish certain features of this condition and lead to possible explanations of the disorder.

1. Premature infants tend to have a positive iron balance when fed according to the method commonly employed.

2. The pigment excretion is not excessive.

3. The anemia affects the red blood cells and hemoglobin equally, develops in spite of treatment, and improves spontaneously.

4. The anemia appears to be due to failure to utilize the available supplies rather than to lack of the substances which compose hemoglobin.

5. An etiologic hypothesis which would explain many of the observed phenomena portrays failure of utilization of the substances which compose hemoglobin due to inadequate medullary space coincident with the premature cessation of hematopoietic activity in the liver.

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OSTEOMALACIA

Report of a Case Treated by Large Doses of Viosterol and Quartz Lamp Therapy Without Improvement

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ON SEPTEMBER 26, 1928, a ten-year-old boy with multiple skeletal deformities was referred to the dispensary of the Northwestern University Medical School because of his inability to walk without crutches or assistance. His appearance at that time is shown in Figs. 1 and 2. A diagnosis of osteomalacia was made, and more or less continual treatment was maintained until June, 1931, when death occurred from nephrosis. Despite the use of large doses of viosterol and quartz light therapy no improvement in the calcification of the bones took place during this time.

The patient was born at term with a birth weight of 8 pounds. The mother's diet had been general without milk. The baby was nursed exclusively until eleven months of age, except for orange juice at six to seven months. No cod liver oil was given. At weaning the child refused to drink cow's milk. Teeth developed at five months and the child walked at one year of age.

When two years of age he had a severe cold, following which the abdomen became large and a diarrhea developed which lasted for over a year. He was admitted to the Children's Memorial Hospital in Chicago in September, 1920. A report from this hospital states that at this time he was a well-nourished baby of two years with negative findings, except for an abdominal condition. The abdomen was distended, with generalized dullness except over the stomach and tympani in the right side. There was no spasticity, tenderness, palpable masses, or shifting dullness. A von Pirquet test was negative, but a rise of temperature to 101° followed the subcutaneous injection of O.T. He remained in the hospital until the end of July, 1931, when he became an ambulatory patient. The diagnosis at that time was tuberculous peritonitis.

The mother states that the first time she noticed any deformity was in 1925 when the child was seven years of age and that the deformities began in the legs as far as she could recollect. By the following year the deformities were marked, as he was taken to the Daily News Sanitarium in the summer of 1926, when eight years of age. At that time the following notes were recorded: "Extreme tachycardia and rapid suffocating breathing. Pigeon breast. Kyphosis. Spinal deviation of 2 inches. Left scapula impacted under right. All joints enlarged and long bones curved. All glands enlarged." Ultraviolet ray exposure was started and increased up to fifteen minutes. He was brought to the sanitarium every other day throughout the year 1926-27 and 1927-28 and given quartz light therapy. Various calcium salts were given during this time.

At the time he was first seen at the Northwestern Dispensary in September, 1928, the following notes were made in addition to the history recorded above.

Family History.—Father died of mushroom poisoning. Mother living and well. One brother living and well.

Physical Examination.—Length. Lying length about 47 inches or 119.5 cm. Head, 52 cm. Chest at nipple line 55 cm. Abdomen 54 cm.

A very badly distorted child with wizened old face. Poor nutrition, poor color. Very intelligent.

Skin pale and white. Blood vessels stand out prominently on surface.

Glands. Cervical, axillary and inguinal palpable. Epitrochlears not enlarged.

Eyes normal position. Ocular movements normal. No nystagmus. Pupils regular, react to light and accommodation.

Head proportionately larger than body. Sutures markedly indented. Bosses prominent. Head much larger than face proportionately.

Ears and nose negative.

Teeth many carious, many absent.

Skeletal. Extreme scoliosis with convexity to the left. Considerably kyphosis. Chest, back, clavicles and scapula distorted.

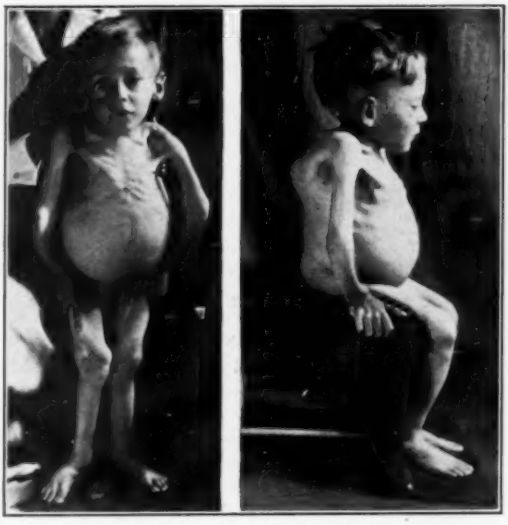

Fig. 1 Fig. 2

Figs. 1 and 2.—Appearance of patient on first observation.

Extremities. Marked bowing of all long bones. Clubbing of fingers and toes.

Heart. Pulse rate 130. No murmurs. Apex beat outside mammary line. Fourth interspace. Border could not be outlined.

Lungs. Râles below constricted portion of lung on right side.

Abdomen. Markedly distended.

Liver. Two fingerbreadths below costal margin and spleen about 3 fingerbreadths. Fluid wave could be elicited.

Blood count. 4,730,000 reds, 40 per cent hemoglobin (Sahli), white count 7,200. Differential: lymphocytes 32 per cent; large mononuclears 1; polymorphonuclears 65 per cent; basophiles 2 per cent.

Urine. Negative.

Intradermal tuberculin test with 8 mgm. tuberculin negative.

Stool. Negative for fat parasites or parasitic ova, and mucus. Consistency, watery; color, yellow.

Roentgenologic report by W. T. Bronson:

There is a marked decalcification of bones rayed. This decalcification includes all long and flat bones, there is a marked bowing of the long bones, irrespective of weight bearing. There are pathologic fractures in the radius and ulna of both

Fig. 3 Fig. 4

Figs. 3 and 4.—Roentgen ray plates when first seen, September 26, 1928.

Fig. 5 Fig. 6

Fig. 5.—March 6, 1929, five months later, after 9 to 15 drops acterol daily (100D viosterol in oil).

Fig. 6.—February 12, 1930, eleven and one-half months after Fig. 5, after 15 to 40 drops of acterol daily.

arms and in both fibulae. The ends of the shafts of some of the long bones show the characteristic saucer-shaped absorption of rickets. The bone changes are suggestive of an extremely severe case of rickets. (Figs. 3 and 4.)

The mother refused to allow the patient to be admitted to the hospital for study or treatment. He was therefore followed and treated in the dispensary until February 6, 1931—a period of two years and five months.

When first seen, September 26, 1928, a blood calcium phosphorous determination was made with the following findings:

Calcium	9.9 mg. per 100 c.c. serum
Phosphorus	2.6 mg. per 100 c.c. serum
Product	25.74

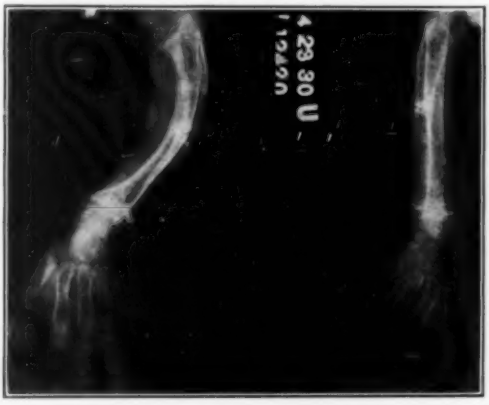

Fig. 7.—April 23, 1930, two and one-half months after Fig. 6, after 40 drops of acterol for four and one-half months. Beginning of use of 40 drops daily of 2500 D viosterol in oil.

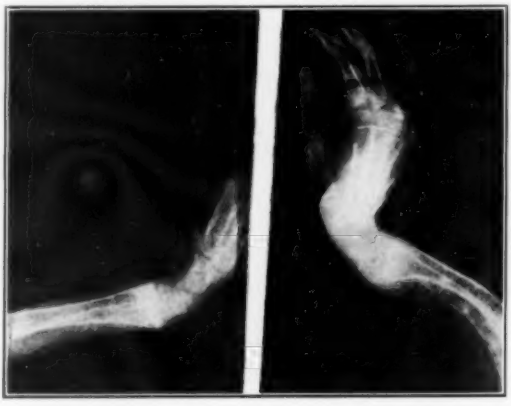

Fig. 8.—February 6, 1931, nine months later, after 40 drops daily for six months and 80 drops for three months of 2500 D viosterol.

TREATMENT AND COURSE

An attempt was made to get the child to take milk by cooking it with cereal, adding flavoring substances, etc. Calcium gluconate (gr. x daily) was given until 10 ounces of milk daily was being taken. This amount was not reached until October, 1929. A general diet of fruit, meat, cereals, leafy and coarse vegetables was ordered. Cod liver oil, 3 teaspoonfuls daily, and 6 drops of acterol were ordered. It was reported as impossible to get the child to take the cod liver oil, so that acterol was increased to 9 drops daily. In February, 1929, the dose was increased to 15 drops daily and again in October to 30 drops. From November 13, 1929, to April 23, 1930, the daily dose was 40 drops. During this time the blood chemistry showed an increasing calcium loss rather than improvement.

Examination Dec. 5, 1928		*May 5, 1929*	
Calcium	8.28	Calcium	6.8
Phosphorus	2.5	Phosphorus	2.14
Product	20.575	Product	14.55

X-ray examination likewise failed to show improvement. A report April 23, 1930, by Dr. James J. Case: "Comparison of the films with those first made shows, I think, a little more calcification. The cortex seems more dense and a little thicker but very little. The old fracture . . . is still incompletely healed. . . . There does not seem to be any progress in the formation of carpal bones—only four are present."

At this time, April 23, 1932, a change was made to 40 drops of 2500 D viosterol. A roentgenologic examination October 10, six months later, showed no improvement despite the fact that in addition to this large dose the child had been exposed to the sun during the summer. The daily dosage was increased at this time to 80 drops (approximately 40 minim or equivalent to 800 drops of 250 D), which was continued until the patient died in June, 1931. A roentgenogram report in February, 1931, reads as follows: "There is no increase in the general calcification of the bones."

On May 29, 1931, the mother reported that following a severe cold, swelling of the legs had developed. Hospital treatment was advised but refused. On June 12 he was brought to the hospital in a moribund condition with marked general edema, and he died 38 hours later. Autopsy refused.

COMMENT

From the history and clinical picture a diagnosis of osteomalacia or late rickets may be made. Wilson[1] and V. E. Pelly[2] from their study of 152 cases in India consider the two conditions as identical, the symptomatologic differences being due to the age of onset rather than pathology.

The etiology can only be conjectured. There are two points in the

history which may be of importance. First the diarrhea with abdominal enlargement when the child was two years of age and which lasted for a year, and, secondly, the unbalanced diet of the child. Holt, Courtney and Fales have shown a definite relationship between fat excretion and calcium metabolism, and late rickets has followed celic disease. The British physicians in India stress the importance of an unbalanced diet as a cause of osteomalacia. From the time of weaning practically no milk and very few vegetables were taken.

CONCLUSIONS

An unusual case of decalcification of the bones, probably osteomalacia or late rickets is presented. The long-continued use of ultraviolet ray (quartz lamp), followed by the use of viosterol in large doses over a period of many months failed to produce any improvement.

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SINO-AURICULAR HEART-BLOCK IN CHILDHOOD

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SINO-AURICULAR block is an irregularity of the heart caused by an intermittence of the rhythmic contractions of the auricle. One or more beats may be lost during a single intermittence. When a single beat is lost, the length of the pause is approximately equivalent to the length of two normal cycles. The condition was first recognized as an entity by Wenkebach[1] in 1907. Rather complete descriptions of this irregularity are given by Lewis[2] and by Wenkebach and Winterberg.[3]

The mechanism responsible for this disturbance has been discussed recently by Resnick,[4] and by Martini and Müller.[5] While there is considerable controversy over the nature of the disturbed physiology in this condition, it is known that vagus inhibition plays an important rôle in most cases. This inhibition may be due to an exaggeration of the normal physiologic function of the vagus, or it may result from other factors acting upon the nerve and stimulating it to a greater activity. In either case the result is an abnormal suppression or transient abolition of the automaticity of the sino-auricular node, or a blocking o fthe impulse before it reaches the auricle. This suppression or blocking then may be said to be caused by either a physiologic or a pathologic hyper-vagotonia. When the node itself is responsible for its absence of activity, the block is also of a pathologic nature; but cases of this type are rare.

The primary etiologic factors which give rise to pathologic forms of sino-auricular block are essentially the same as those which produce auriculoventricular block. In general they may be classified into two main groups, infectious and toxic. Organic lesions of the heart are a frequent cause of auriculoventricular block, but they are seldom responsible for an arrest of the sino-auricular node. This is particularly true in children.

Digitalis is the most important of the toxic causes. In Levine's[6] review of the literature he pointed out that digitalis was the most frequent cause of sino-auricular block. Schwartz and Schwedel[7] noted this type of block in three of the twelve children on whom they were making digitalis studies. Other drugs may induce a similar toxic effect. Cushny[8] found this type of block in dogs following the administration of aconite. Eyster and Meek,[9] and Cohn[10] report its occur-

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rence in experiments with morphine. Heimann[11] reported a case in which the irregularity disappeared after thyroidectomy.

Of the infections, rheumatic fever is the most common cause of sino-auricular block; however, it may be provoked by any acute infection. Hyman[12] and Parsonnet[13] have reported cases occurring in influenza, and Poulton[14] has reported a case following pneumonia. Smith[15] has stated that, "In diphtheria this irregularity (sino-auricular block) was invariably observed in convalescents." While cases of this type of block do occur in the course of and during convalescence from diphtheria (Marvin[16] and Reid[17]), Smith's impressions are not shared by other observers. Halstead[18] made an intensive electrocardiographic study of 100 cases of diphtheria and never noted this condition. In a similar study of 50 consecutive cases, Shookhoff and Taran[19] found a sinus-arrhythmia in 56 per cent but observed no cases of sino-auricular block. In Marvin's[16] series of 90 cases the block was present in only one instance.

The physiologic group includes those cases in which the irregularity is due to a hypervagotonia of unknown cause. White's[20] case of a twenty-four-year-old athlete in whom the block occurred after exercise is an example of this type. Carter[21] and Cuervo[22] point out that occasionally in athletes there is found sudden halving and doubling of the pulse rates and that these abrupt changes are due to sino-auricular block. Wedd and Wilson[23] report a case of "auricular standstill" in a twenty-four-year-old male whose vagotonia they attributed to a psychogenic factor. Gallavardin[24] reported a case in a fifty-year-old male adult who exhibited the block only when he was in the upright position. In one of the earlier papers on this subject, Eyster and Evans[25] report the case of a thirty-one-year-old male who had had a slow irregular pulse for many years and who was otherwise in excellent health. They concluded from their studies on this patient that the block resulted solely from vagal activity.

REPORT OF CASE

Because the recorded cases of sino-auricular block are rare and especially so in children it was felt that a report of this case would be of value.

L. T., aged six years, was brought to the clinic because his mother noted that he had a tachycardia. He had never complained of any rheumatic symptoms, and as far as could be determined he had sustained no recent infections.

Physical examination revealed a moderately well-nourished child who was in apparently good health. The findings except those associated with the heart were essentially negative. There was a grossly discernible rhythmic variation in the pulse rate which was dependent upon his respiratory rhythm, but in addition to this there were numerous sudden changes in rhythm which were independent of respira-

TABLE I

DATE	ELECTROCARDIOGRAMS						CLINICAL OBSERVATIONS			ATROPINE
	CURVE NO.	RATE	P.R.	P.P.	P′P′	ABNORMAL P-WAVES	RATE	IRREGULARITY	OCULOCARDIAC REFLEX	
7/16/30	I	103	0.14 0.16	0.50	1.02	x	140	xx	xx	H—Subcutaneous M—By Mouth
9/ 2/30	II	100	0.16 0.18	0.58	0	0	96	0	x	
6/15/31	III	103	0.14 0.16	0.50	0	x	86	xx		
6/29/31	IV	75?	0.16	0.54	1.12	x				
8/31/31	V	120	0.16	0.48	0	x	110	xx		
12/28/31	VI	72?	0.16	0.66	1.26	0	84	x		
3/28/32	VII	115	0.14 0.16	0.48	0	x	110	xx		
6/28/32	VIII	119	0.16	0.48	1.12	x	96 102	xx	xx	$\frac{3}{500}$ M
7/13/32	IX	111	0.16 0.18	0.52	0	0	110 131	xx	xx	$\frac{1}{100}$ H
7/22/32	X	120	0.16	0.48	0	0	126	0	0	$\frac{1}{30}$ H
7/25/32	XI	100	0.16	0.60	1.36	x	115	xx	xx	0

The blocked cycles (P′P′) are all at least twice as long as the shortest normal cycle (P.P.).

tions. There was a soft systolic murmur at the apex which was not transmitted beyond that point. The area of cardiac dullness was not enlarged. Teleroentgenogram revealed a heart shadow of normal size and shape. Routine urinalyses and blood counts were normal. Electrocardiogram portrayed typical sino-auricular block.

He was put on complete bed rest for several weeks. His temperature remained normal and the irregularity persisted.

In the course of the next year there were no changes. During this time the pulse rate varied between 78 and 140. The block being present at both the higher

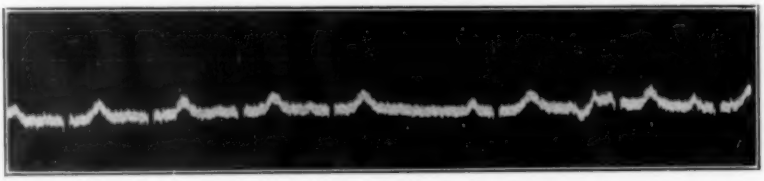

Fig. 1.—Lead II. Portion of Curve I. The fifth auricular cycle is blocked.

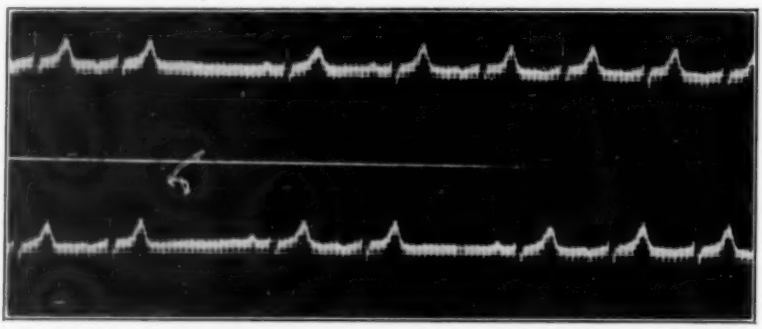

Fig. 2.—Curve IV. Both strips are recordings of Lead I. The P-waves are inverted except following the three blocked beats.

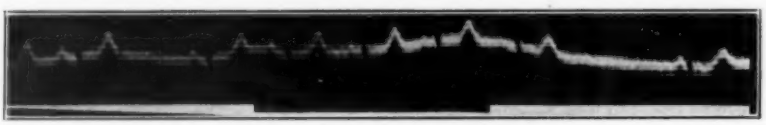

Fig. 3.—Curve XI. Lead II. Nodal escape after last blocked beat.

and lower pulse rate levels. The systolic murmur gradually disappeared. He gained weight, attended school and led the life of a healthy normal child.

On June 27, 1932, he was admitted to the hospital for further study. Atropine was administered in slowly increasing doses. The irregularity did not completely disappear until the twenty-third day of medication when he was receiving $\frac{1}{30}$ gr. of atropine sulphate subcutaneously four times a day. This also was the first time that ocular pressure was without effect. By the following day the signs of atropinization disappeared and the arrhythmia returned.

The patient has been under our observation for thirty months, and now at the age of eight years the sino-auricular block is still present.

DISCUSSION OF ELECTROCARDIOGRAMS

Only significant portions of the electrocardiographic curves are reproduced here. The important features of all the curves and the correlated clinical findings are tabulated in Table I. Evidences of block were not apparent in four of the curves (Numbers III, V, VII, and IX), but clinical evidence of the irregularity was present on the same day the curve was taken. The measurements of the longest disturbed cardiac cycles are tabulated under P′P′ and the duration of the shortest cycles are tabulated under P.P. It is to be noted that the longer cycles (P′P′) are all at least twice as long as the corresponding normal P.P. cycle.

Several of the curves showed changes in the shape and phase of the P-waves. Wilson[26] attributes this type of aberration to a vagal influence which induces a shifting of the pacemaker. In Fig. 2 the P-wave is inverted indicating an ectopic focus of the pacemaker. Immediately after a blocked beat it becomes upright, suggesting that the node has recovered.

When atropinization was complete, the electrocardiograms (Curve X) were normal. The P-waves were all upright, the rate was accelerated to 120, there were no blocked beats, ocular and vagal pressure, and breath holding were without effect.

Fig. 3 is a portion of Curve XI which was taken two days after atropine was discontinued. The dropped beat in this curve is followed by a nodal escape, an occurrence not uncommon in cases of sino-auricular block. In another portion of this curve (not reproduced here) there is an auricular standstill of 3.28 seconds, resulting from ocular pressure.

COMMENT

The sino-auricular block persisted in this patient until he received a total daily dose of $\frac{1}{30}$ gr. of atropine sulphate subcutaneously. It was at this time only that the oculocardiac reflex could not be obtained. The relationship of the disappearance of the block to atropine, the associated marked sinus arrhythmia, and the absence of any other etiologic factors indicate that an abnormal exaggeration of the physiologic activity of the vagus is the immediate and probably solitary cause of the irregularity. This conclusion is in accordance with the views of Resnick[4] who believes that "sino-auricular standstill" results from vagal influences on impulse formation in the sino-auricular node as does also sinus arrhythmia. He says that "sino-auricular standstill is merely a special form of sinus arrhythmia." The association of the two conditions in this patient and the apparent benignity of the process, in spite of its chronicity, are, at least in this instance, confirmation of Resnick's point of view.

REVIEW OF THE LITERATURE

Since Levine's[9] review of the literature in 1915 the number of reported cases of sino-auricular block has more than quadrupled. Yet in spite of this increasing literature the condition remains a clinical rarity in childhood. The cases in this age group thus far reported are summarized in Table II. A review of these cases is merited for they have never been presented or considered as a collective clinical group.

In his paper on circulatory disturbances of childhood Wilson[27] stated that he had never observed a case of sino-auricular block in a child.

The first report of a juvenile case was made by Joachim.[28] His patient was fifteen years old, and had acute rheumatic fever, pericarditis,

TABLE II

REPORTED CASES OF SINO-AURICULAR BLOCK IN CHILDHOOD

AUTHOR	AGE	DISEASE	P.P.	P'P'	DURATION
Joachim	15	Pericarditis digitalis	0.60	1.20	Several days
Brown	11	Acute rheumatic fever	0.64	1.6	9 months
Smith	5	3 months after diphtheria	0.52	0.88	Not stated
	4	During diphtheria	0.48	0.90	
	8	Convalescent diphtheria	0.56	0.96	
	6	Convalescent diphtheria	0.56	1.0	
	6	Mumps	0.64	1.02	
	4	Pertussis	0.64	0.92	
Reid	10	Diphtheria 5 years ago	Little or no auric. activity		5 years
Schwartz Schwedel	3 cases	Digitalis	No curves given		22 hours
Svejcar Helbichova	At birth	Congenital heart	0.40	1.10	Died at 5 months
Author	6	No disease	0.48	1.12	30 months

P. P.—Duration in seconds of shortest cardiac cycle.
P'P'—Duration in seconds of the cycle which includes a blocked beat.

and was apparently decompensated. The cardiac irregularity followed digitalization and did not completely disappear until several days after digitalis had been discontinued. This case falls into that group of cases in which the block is due to toxic causes.

In Levine's review of the literature he was able to collect 14 cases and to add 4 of his own. Joachim's case was the only child included in this group.

In 1927 Barlow[29] reviewed the literature for the period following Levine's report. He collected 32 cases and added 4 of his own. The only children included in this group are six cases reported by Smith[30] and one by Brown.[31]

Smith was also of the opinion that sino-auricular block is in all likelihood a physiologic manifestation and not a pathologic condition. His cases all followed acute infections, and his youngest patient was four years old. His curves, however, do not completely meet the re-

quirements for the interpretation of sino-auricular block, as they fall considerably short of having the disturbed cycles approximately equal to two normal cycles. Some of those which are designated as being examples of sino-auricular block could certainly be interpreted as instances of marked sinus-arrhythmia. He does point out a relationship between the two conditions and emphasizes the frequent difficulty of distinguishing them at times. His cases were not followed to determine the duration of the arrhythmia.

Brown's[31] patient was an eleven-year-old child in whom the block was first discovered during an attack of acute rheumatic fever. His heart was enlarged and he probably had a mitral lesion. The block could be temporarily abolished by atropine. After an interval of nine months another electrocardiogram showed that the block was still present. The case was not followed any farther. In this instance the assumption is permissible that the block was due to vagal influences. Whether the rheumatic infection induced a higher degree of susceptibility of the nodal tissue to the vagus, or whether the vagus was simply more active, is problematical.

Reid's[17] case is the only example of permanent cardiac damage. His patient was a ten-year-old boy in whom the block appeared at the age of five years while he was convalescing from diphtheria. Electrocardiographic tracings showed little or no activity of the sino-auricular node. Aside from the bradycardia the heart was negative and the child enjoyed good health. This case is most likely an example of severe injury of the sinus nodal tissue.

The cases of sino-auricular block reported by Schwartz and Schwedel[7] are incidentally mentioned by them as another type of digitalis effect. Individual case records are not presented. The block occurred on about the seventh day of digitalis administration and lasted approximately twenty-two hours.

Congenital heart-block is an extremely rare condition. Nicolson, Shulman and Green[32] collected 22 cases from the literature; all of these were of the auriculoventricular type. The only case of congenital sino-auricular block I have been able to find in the literature is one reported by Svejcan and Francova-Helbichova.[33] Their patient had other cardiac anomalies and died at the age of five months.

Wood's[34] report of an eleven-year-old boy with ventricular escape might also be included in this group of cases under review. In this case there was some depression of the sino-auricular node with the escape of an ectopic focus. The arrhythmia was transient, and it was thought to be due to the active rheumatic infection which the patient had at the time of the observation. The immediate cause is attributed to the vagus.

The author's case is the only one in the group for which an etiologic factor such as infection or intoxication could not be found. Mention has already been made of several cases of this type that have been reported in adults. If a primary vagotonia is provocative of sino-auricular block, one would expect it to occur more frequently in childhood, when sinus arrhythmia which is a physiologic expression of vagotonia is so common. Sinus arrhythmia is recognized as a normal finding in children. Paton[35] considers it an almost constant finding; Lincoln[36] records an incidence of 86 per cent in 325 children from two to thirteen years of age; and Shookhoff and Taran[19] found it present in 78 per cent of their group of normal children.

In itself sino-auricular block seems to be a benign process, and it is usually a transient phenomenon. It can be distinguished from certain other more serious forms of cardiac irregularity only by means of electrocardiographic studies. Occasionally, as it was in my case, it may continue over a long period of time in the absence of any cardiac findings and without apparent detriment to the well-being of the patient. Gallavardin[24] reported a similar case in an adult in whom the block persisted for nine years. The case reported by Reid[17] is an example of probable intracardiac injury with permanent abolition of the functioning of the sino-auricular node, but even in this case the patient's health was unaffected.

Because of the nature of sino-auricular block, treatment is usually not indicated except where it can be directed against the underlying causes. The employment of atropine is of value in demonstrating the part played by the vagus in the production of this phenomenon, and for the abolition of symptoms which occasionally occur. So few cases of more or less permanent block have been observed over a long period of time that accurate prognostic information concerning them is not available. In the absence of such information it would seem advisable to make frequent clinical and electrocardiographic observations on these patients, even though in most instances the block is apparently of a benign nature.

SUMMARY AND CONCLUSION

A case of sino-auricular heart block in a child is presented, and the literature on this type of cardiac irregularity in childhood is reviewed.

The relative infrequency of sino-auricular block in childhood as compared to its incidence in adults is pointed out. The apparent relationship of this condition to sinus arrhythmia suggests that the so-called "physiologic type" of block should occur with greater frequency in children.

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ACUTE POLIOMYELITIS
STUDIES OF STREPTOCOCCI ISOLATED FROM THROATS AND RAW MILK IN RELATION TO ONE EPIDEMIC

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THE epidemic studied occurred at White Bear Lake, Minnesota, a town of about 2000 inhabitants. The first case of poliomyelitis occurred September 20, 1931; the second case, October 4, and the third case, October 5. Little attention was paid to the occurrence of these cases, but when four occurred, two in each of two families about October 10, the townspeople became alarmed and demanded that schools be closed, and that other presumably preventive measures be instituted. Our study of the epidemic was begun on the evening of October 12, at the request of the president of the school board, when the throats of several patients and of other members of stricken families were swabbed for cultures and for precipitin tests, and a skin test for susceptibility was made.

Owing to the fact that an institutional outbreak of poliomyelitis had been traced to raw milk,[1] the milk supply was determined and samples for cultures were procured. The following day swabbings were taken from the throat, and a skin test for susceptibility to poliomyelitis was made on each of 104 of the school children presumably exposed in the schoolrooms where three cases had occurred a short time before. The precipitin test, made with the cleared nasopharyngeal washings and the poliomyelitis antistreptococcus serum, and studies of cataphoretic velocity of the primary cultures in glucose-brain broth, determined October 14, revealed the presence of the "poliomyelitis" streptococcus in the throats of each of the patients whose throats were swabbed, in the majority of the contacts in stricken families, and in 29 per cent of the 104 children from the three schoolrooms where three cases had occurred. Studies of the cataphoretic velocity, and other studies, revealed the presence of large numbers of the "poliomyelitis" streptococcus in several samples of raw milk obtained from the two homes in each of which two cases had occurred and in smaller numbers in each of five other samples of raw milk. This streptococcus was proved absent in four samples of pasteurized milk from which cultures were

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made as controls, including the brand used in the home where the first case occurred. On the basis of the relatively low incidence of the streptococcus in the throats of the children (29 per cent) exposed, as compared with contacts in more severe outbreaks (60 to 80 per cent),[4, 5] because of the lateness of the season and results obtained previously in an institutional outbreak apparently due to milk, we reported to the Health Officer, Dr. A. E. Voges, on the night of October 14, that a severe outbreak was not likely to occur, that the schools should be kept open, and advised that a close watch be kept by the teachers, school nurse, and parents, especially of children who reacted positively to the skin test and hence were presumably susceptible, and who were proved to be carriers of the streptococcus by a positive precipitin reaction. Also, it was recommended that the family physician be called for early diagnosis and serum treatment if illness occurred, and that all raw milk be scalded, pasteurized or boiled. Cooperation by the school, health authorities, and public was splendid, and the people generally became assured. However, owing to the controversy regarding the comparative value of pasteurized and raw milk, which were used about equally in the town, the advice regarding heating of milk was not generally followed, and presumably in consequence, judging by the results of further study of raw milk, cases continued to occur.

On the basis of these results, and considering the willingness of the health officer, school authorities, and people generally to cooperate, it was decided to make a more complete survey. Accordingly, the throats of all school children, including the Catholic parochial school, from kindergarten through high school, were swabbed; a skin test of nearly all was made October 19; and a study of the relation of the milk supply to the epidemic was continued. The results of the skin test, presumably indicating susceptibility, were summarized and reported in the afternoon, and the results of the precipitin test, indicating the carrier state of the streptococcus, on the evening of the following day.

Through the advice given following the preliminary survey, and the continued splendid cooperation of the school authorities, diagnoses were made early in the cases that occurred subsequently; and treatment, either with convalescent serum or poliomyelitis antistreptococcus serum, was promptly instituted, resulting seemingly in reduction in the incidence and degree of paralysis.

THE PRECIPITIN REACTION

The precipitin reaction in this study, as in previous epidemics,[4, 5, 7] was made by layering a small amount of the centrifuged, cleared suspension in gelatin-Locke solution (2 c.c.) of the material obtained on the swabs from the nasopharynx, over the poliomyelitis antistrepto-

coccus serum in narrow tubes (0.5 by 3 cm.). These were incubated for two hours and then read. A cloud at the interphase, a positive reaction, indicated the presence of the streptococcus with which the serum was prepared. The antistreptococcus serum was prepared in the horse by repeated intravenous injections, during many months, of the killed streptococcus, freshly isolated from patients with poliomyelitis, preserved during the period of immunization in dense suspension of glycerin (2 parts) and 25 per cent sodium chloride solution (1 part). Controls with normal horse serum, and other antistreptococcus serums, were not used because of the large number of swabbings to be tested and because these serums had been used previously with negative results in a large number of similar tests.

The results of the precipitin reaction in different groups tested a week apart are summarized in Table I. It will be noted that the incidence of positive reactions was about equal (29 per cent and 25 per

TABLE I

RESULTS OF THE PRECIPITIN REACTION OF DIFFERENT GROUPS OF PUPILS ACCORDING TO DATE

GROUPS	DATE, OCTOBER	PERSONS WHOSE REACTIONS WERE DETERMINED	POSITIVE	
			PERSONS	PER CENT
Persons from schoolrooms where cases had occurred	13	104	30	29.0
Miscellaneous contacts	13	20	5	25.0
All schools	19	965	72	7.4
Miscellaneous	20	22	2	9.0
Total		1,111	109	9.8

cent) in the two groups swabbed October 13, and about three times as high as in the two groups swabbed seven and eight days later (7.4 per cent and 9 per cent, respectively). Groups 1 and 3 are not comparable as to numbers, age, residence, and exposure, but groups 2 and 4 are comparable in these respects.

The cataphoretic velocity of the streptococci from the nasopharynx that grew out overnight in cultures in glucose-brain broth was determined in 101 of the persons of whose throats swabbings were made October 13. The velocity of the streptococci from each of the thirty persons who yielded a positive precipitin reaction with the poliomyelitis antistreptococcus serum, and from six others, was chiefly "poliomyelitic" (36 per cent). Three of the thirty-six persons who yielded "poliomyelitic" streptococci had mild infections of the upper part of the respiratory tract when swabbed. The velocity was that characteristic of streptococci from normal persons in forty-four instances, and none of the individuals had colds when swabbings were made. In thirteen nasopharyngeal cultures the velocity was that of streptococci isolated in colds, and ten of the patients from whom these cultures

were obtained were having colds, and three were in normal condition when swabbings were made. In five cases the velocity of the organisms was that of streptococci isolated in cases of influenza, and of these, three patients were having infections of the upper part of the respiratory tract, whereas two were in normal condition. The velocity of organisms obtained from two patients was "arthrotropic"; neither had a cold at the time.

The results of the precipitin reaction, according to schools, are summarized in Table II. No cases had occurred in the high school, Webster School, or St. Mary's School. The incidence of positive precipitin reaction in these schools was 5.6, 4.4 and 5.0 per cent, respectively. At the Washington School, where three cases had occurred, the incidence of positive precipitin reactions was 12.3 per cent. A chocolate flavored, unpasteurized milk was dispensed in considerable amount from half pint bottles at this school, but not at the other schools. Cultures

TABLE II

RESULTS OF PRECIPITIN REACTION OF PUPILS ACCORDING TO SCHOOLS, OCTOBER 19, 1931

SCHOOL	PERSONS WHOSE REACTIONS WERE DETERMINED	POSITIVE	
		PERSONS	PER CENT
High	320	18	5.6
Washington	276	34	12.3
Webster	90	4	4.4
St. Mary's	279	16	5.0
Total	965	72	7.2

from two samples of this milk, obtained a week apart, yielded the "poliomyelitis" streptococcus, which proved to have "neurotropic" virulence in rabbits and mice.

THE SKIN TEST FOR SUSCEPTIBILITY TO POLIOMYELITIS

In a previous study, it was shown that the incidence of positive skin tests, with poliomyelitic streptococcus antigens, in different age groups was roughly proportional to the incidence of susceptibility to poliomyelitis.[6] The antigen used in this study was a heat-killed suspension of a mixture of three strains of streptococci isolated from the spinal fluid of three Macacus rhesus monkeys as they developed poliomyelitis following intracerebral injection of "sterile" filtrates of poliomyelitic virus. The three strains of streptococci represented two highly virulent viruses, one isolated in a fatal case of poliomyelitis in a child; the virus was in the fourth passage in the monkey. The other virus had gone through years of more or less continuous, successive passages through monkeys, and was kindly sent to us by Dr. Paul F. Clark, of the University of Wisconsin. The streptococci from the former virus represented one-third of the vaccine and was in the thirty-fourth

rapidly repeated subculture in glucose-brain broth. The two strains from the latter virus represented one-third each of the vaccine, respectively in the sixteenth and fifth rapidly repeated subcultures in glucose-brain broth. The three strains were grown separately for one culture generation in large amounts of 0.2 per cent dextrose broth for the immunization of horses, and the organisms, harvested with a continuous feed centrifuge, were suspended in glycerin (2 parts) and 25 per cent sodium chloride solution (1 part) so that each cubic centimeter of this suspension contained approximately 1,000,000,000,000 streptococci and had been preserved in the refrigerator for two years. The characteristic cataphoretic velocity which the streptococcus possessed, as filed away on isolation, was still at hand two years later, when dilutions were made in saline solution, so that 1 c.c. contained approximately 2,000,000,000 organisms. This diluted suspension was heated to 75° C. for one hour, and then diluted further with nine parts of physiologic saline solution containing 0.2 per cent phenol. In making the test, approximately 0.05 c.c., or 40,000,000 organisms, were injected intradermally on the flexor surface of the left forearm, with a fine (26 gauge) needle. Readings were made approximately twenty-four hours later. The results were recorded according to the arbitrary scale of 0 to 4+, 0 representing no redness, 1 representing redness only in the area blistered, or about 0.5 cm. in diameter, 2 representing redness from 0.5 to 1 cm. in diameter, 3 indicating redness 1 to 2 cm. in diameter, and 4 meaning redness more than 2 cm. in diameter. The control injection of saline solution, containing 0.2 per cent phenol, was omitted, because in a large number of persons tested previously, it almost never caused any reaction and in the very few instances when it did, the reaction was always slight, graded never more than a 1+. Owing to lack of knowledge regarding the harmlessness of the skin test, or for other reasons, some parents objected to having the test made. Their request was heeded in all instances when it was made known to us by the children. In a few instances the children forgot to tell us of their parents' wishes, or purposely withheld the information, and the test was inadvertently given as the throat was swabbed, in a routine manner.

The results obtained in the different grades, and hence according to the approximate age, are summarized in Table III. It will be seen that with the increase in age the incidence of negative reactions increased and the incidence of positive reactions decreased correspondingly. Thus, of forty-six children from two months to five years of age (preschool and kindergarten) 28 per cent reacted negatively, 11 per cent reacted moderately positively, and 61 per cent reacted strongly positively, a total of positive reactions of 72 per cent. Of 325 children from six to nine years of age, in school grades 1 to 4, 28 per cent re-

acted negatively, 27 per cent moderately positively, and 44 per cent strongly positively, a total of positive reactions in 72 per cent. Of 195 children who were in the fifth, sixth, seventh and eighth grades, and who were about ten to thirteen years of age, 32 per cent reacted negatively, 26 per cent, moderately positively, and 42 per cent, strongly positively, or a total of 68 per cent positive reactions. Of 182 high school students who were from fourteen to seventeen years of age, 55 per cent reacted negatively, 18 per cent, moderately positively, and 26 per cent, strongly positively, or a total of 44 per cent positive reactions. Of twenty teachers who volunteered for the test, 75 per cent reacted negatively, 5 per cent, moderately positively, and 20 per cent, strongly positively, a total of 25 per cent of positive reactors. These figures are in sharp contrast to the results obtained with this same

TABLE III

RESULTS OF SKIN TEST FOR SUSCEPTIBILITY TO POLIOMYELITIS

GROUPS OF PUPILS AND TEACHERS	PERSONS WHOSE REACTIONS WERE DETERMINED	PER CENT NEGATIVE (IMMUNE) 0 AND 1+	PER CENT POSITIVE (SUSCEPTIBLE)		
			MODERATELY 2+	STRONGLY 3 AND 4+	TOTAL 2, 3, 4+
Preschool and kindergarten, 2 months to 5 years	46	28	11	61	72
Grades 1 to 4, 6 to 9 years	325	28	27	44	72
Grades 5 to 8, 10 to 13 years	195	32	26	42	68
High school, 14 to 17 years	182	55	18	26	44
Teachers, 20 years +	20	75	5	20	25
Persons having had poliomyelitis, ages 3 to 21 years	44	89	11	0	11

antigen in forty-four persons, mostly children, who had had poliomyelitis. Of these, 89 per cent reacted negatively, and 11 per cent moderately positively. None reacted strongly positively.

The skin test was made twice, a week apart, on thirty-five children. Of these, on the second test, ten reacted identically as in the first test, ten reacted slightly less, and five reacted markedly less; ten reacted slightly more positively during the second test. In other words, the skin reaction in the second test was essentially like that in the first in all but five of the thirty-five children tested, and in each of the five it had become markedly less positive. The precipitin test was positive in the first test in each of three of the five children whose skin reacted markedly less to the second test. It was not made in the other two. Four of the five children had had mild symptoms, resembling in one or more respects those of abortive poliomyelitis shortly before, or at the time of, the first test, and had recovered before the second test was made.

Each of twenty-two persons was subjected twice to the precipitin and skin tests. Of these, ten reacted positively to the first precipitin test, and only one, who was negative in the first series, reacted positively in the second series. Of the eleven children who reacted positively to the precipitin test, eight (73 per cent) were users of raw milk and three of pasturized milk. Of the eleven who reacted negatively only five (45 per cent) were users of raw milk and six of pasteurized milk.

Since poliomyelitis occurs more commonly among males than females (twelve males and nine females in this epidemic), the results of the skin reactions were studied according to sex, and recorded on the arbitrary scale of 0 to 4+ in 714 cases. Of the 360 males tested, forty (11 per cent) yielded a completely negative reaction; eighty-seven (24 per cent) a reaction graded 1+; eighty-six (24 per cent), a reaction graded 2+; 109 (30 per cent), a reaction graded 3+, and thirty-eight (10 per cent), a reaction graded 4+. Of 354 females, sixty-three (18 per cent), yielded completely negative reactions; eighty (23 per cent), a reaction graded 1+; eighty-nine (25 per cent), a reaction graded 2+; ninety-six (27 per cent), a reaction graded 3+, and twenty-six (7 per cent), a reaction graded 4+. In other words, completely negative reactions occurred in 11 per cent of the males in contrast to 18 per cent of the females. Slight (graded 1+) and moderate (graded 2+) reactions were alike in the two sexes, whereas strongly positive reactions (graded 3 and 4+) occurred in 41 per cent of males in contrast to 34 per cent of females.

During the reading of the tests we became impressed by the uniformity of the reaction in different members of some of the same families. Hence, the results of the skin reaction were studied according to family groups. Altogether, the children of school age in 146 families were represented. The reactions were identical, or varied only slightly among the different members in eighty-nine families; and of these eighty-nine families the reaction of each member was consistently slight or absent in twenty-four, moderately positive in twenty-five, and strongly positive in forty. In the remaining fifty-seven families, the reactions of different members varied greatly, often from grade 0 to 4+.

THE RELATION OF THE MILK SUPPLY

The epidemic was peculiar in that the incidence of multiple cases in family groups was unusually high (two each in three families and three in one), and in that the individual cases in each of these stricken families occurred almost simultaneously: on the same day in one, one day apart in two of the three families in which two cases in each occurred, and two days apart in the family in which three cases oc-

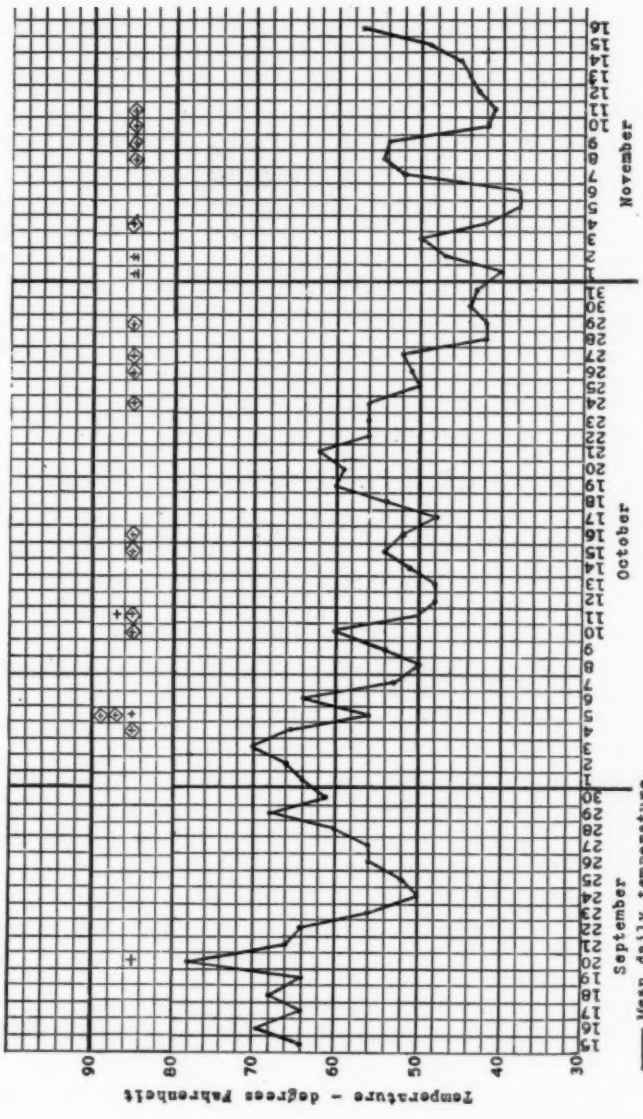

Fig. 1.—Curve of the local atmospheric temperature of the weather in relation to the occurrence of cases of poliomyelitis.

curred, just as if the source of infection for each was a common one and not due to exposure to the earlier case, as is usual. The milk used by each of these four families was a raw product in which the "poliomyelitis" streptococcus was demonstrated. Also, the cases were widely scattered, usually without exposure to other cases, and the

cases occurred explosively in family and other groups, with a considerable interval of time between each two groups. Thus, the first case occurred September 20, nine cases occurred between October 4 and 15, seven between October 24 and November 4, and four between November 8 and 11 (Fig. 1). Three cases occurred on the same day, October 5; all of the three patients were users of raw milk from which the streptococcus was isolated. The epidemic was peculiar, further, in that cases continued to occur later into the autumn than is usual, after the general prevalence of the "poliomyelitis" streptococcus in the throats of children had largely disappeared (the incidence of its isolation being 29 and 7.4 per cent, respectively, in the first and second studies, in contrast to a common incidence of 50 to 70 per cent of the streptococcus in the throats at the height of epidemics in summer[4, 5, 7]). Throughout the period when cases occurred there was no diminution in the incidence of the streptococcus in the raw milk, which was continued to be supplied to about 56 per cent of the population.

The results obtained from study of the relationship of the milk supply to frank and abortive cases, to minor illnesses, and to the presence of the "poliomyelitis" streptococcus in the throats, are summarized in Table IV. The highest incidence of users of raw milk, as shown, was among those who had frank (76 per cent) and abortive (72 per cent) cases. The next highest incidence of users of raw milk was among each of the groups that gave a positive precipitin reaction: well children (74 per cent), children having had colds or other minor complaints (71 per cent), those giving a history of slight illness at the time of study (67 per cent). Among the children with a history of slight illness as obtained at the time of study, but without reference to the result of the precipitin test, the incidence of users of raw milk was 60 per cent. The incidence of users of raw milk was still lower when all children of all schools were taken together (56 per cent), and lower yet among well children with negative precipitin tests (53 per cent) and among those having had colds and other minor complaints (51 per cent) as reported by the school nurse, without reference to the precipitin test.

It is generally believed that a spell of cold weather at the time of epidemics of poliomyelitis tends to diminish the morbidity rate, and that a return of warm weather in epidemic areas is followed by an increase in the number of cases. Through the cooperation of the St. Paul station of the Weather Bureau, U. S. Department of Agriculture, only twelve miles distant from White Bear Lake, we obtained the mean temperature for the period of the epidemic. The daily temperature and the occurrence of the cases are recorded graphically in Fig. 1. It will be noted that the three waves in the occurrence of cases, exclusive of the first case, occurred coincidental with or shortly after three distinct

periods of a decided rise in temperature of the weather just as though the development of cases were due to inoculations from the milk which came to harbor unusual numbers of the "poliomyelitis" streptococcus because of the warm spell in the weather and the consequent inadequate refrigeration of the milk. In agreement with this probability is the fact that the town's supply of ice became exhausted at about the time the cases occurred in epidemic proportion in October. The back porch served as the ice box for refrigeration of the milk at the homes where cases occurred throughout the course of the epidemic.

TABLE IV

RELATION OF THE MILK SUPPLY TO FRANK AND ABORTIVE CASES, TO SLIGHT ILLNESSES, AND TO THE PRESENCE OF THE "POLIOMYELITIS" STREPTOCOCCUS IN THROATS AS DETERMINED BY THE PRECIPITIN REACTION

GROUPS OF PUPILS	PUPILS	CONSUMERS OF MILK			
		RAW		PASTEURIZED	
		NUMBER	PER CENT	NUMBER	PER CENT
Had poliomyelitis	21	16	76	5	24
Had abortive cases of poliomyelitis	32	23	72	9	28
Had had colds or other minor complaints, as reported by school nurse	78	40	51	38	49
Had had colds or other minor complaints, and gave positive precipitin reaction	7	5	71	2	29
Well, and gave negative precipitin reaction	605	326	53	279	47
Well, and gave positive precipitin reaction	34	25	74	19	26
Gave history of slight illness at time of study	188	114	60	74	40
Gave history of slight illness and positive precipitin reaction	36	24	67	12	33
Of all schools	709	399	56	310	44

CULTURES FROM THROATS AND MILK

Samples of about 10 c.c. each of milk were taken for culture, under sterile conditions, from the usual containers, on October 12, 13 and 20, and on November 4 and 14, and as controls after the epidemic had disappeared, December 10. These were transported to the laboratory at Rochester with as little delay as possible, and cultures were made at once, or after refrigeration overnight, by drawing about 0.4 c.c. of the milk from the bottom of the tube into a pipette with a long capillary end. Several drops were streaked over the surface of blood-agar plates, and with the rest tall tubes of glucose-brain broth were inoculated, and in order to favor the growth of streptococci that grow under partial oxygen tension, most of the inoculum was placed in the bottom of the tube surrounding the pieces of brain. Similar cultures were made from nasopharyngeal swabbings. The cultures were incubated only overnight, at 35° C., in order that the parasitic, relatively

anaerobic streptococci, if present in the milk, might outgrow the saprophytic, aerobic bacteria, as they do in this medium if inoculated with material from open foci of infection, such as tonsils, pyorrheal pockets, and the nasopharynx (Table V). The growth on blood-agar was recorded, and if green-producing colonies of streptococci were at hand, subcultures from single colonies were made in glucose-brain broth, for studies of cataphoretic velocity. Satisfactory cultures from the nasopharynx of 96 of the 104 persons whose throats were swabbed during the first survey were obtained on blood-agar plates. Of these, 88 revealed indifferent or green-producing streptococci; 18, hemolytic staphylococci; 5, staphylococci; 1, Bacillus mucosus, and 1, pneumococcus mucosus. Colonies of Micrococcus catarrhalis were present in varying numbers in most of the cultures.

The results of direct plating of the milk on blood-agar, during and after the epidemic, are summarized in Table V. Hemolytic streptococci were not isolated in a single instance. Staphylococci,

TABLE V

INCIDENCE OF ISOLATION OF GREEN-PRODUCING STREPTOCOCCI FROM MILK

TIME	MILK	BRANDS	SAMPLES	GREEN-PRODUCING STREPTOCOCCUS, BLOOD-AGAR PLATES	
				NUMBER	PER CENT
During epidemic	Raw	11	42	26	62
	Pasteurized	5	21	6	28
After epidemic, December 10, 1931	Raw	10	17	15	88
	Pasteurized	4	4	1	25

in varying but usually small numbers, were frequently isolated. Pure cultures in glucose-brain broth, inoculated from fished single colonies, never revealed poliomyelitic cataphoretic velocity. There was a great preponderance of streptococci, which produced a greenish type of hemolytic, slightly hemolytic, or indifferent colonies on blood-agar plates. As shown in Table V, the incidence of this type of streptococcus was far higher in cultures of raw than of pasteurized milk, both before and after the epidemic. Aside from a slightly larger green zone surrounding the colonies of organisms derived from raw milk during the epidemic, there was no recognizable difference in the character of the colonies of streptococci from the milk, during and after the epidemic. The striking difference between the raw and the pasteurized milk at the time of the epidemic was apparent only in the cataphoretic velocity of organisms from primary and single colony cultures. The raw milk at the time of the epidemic yielded streptococci having chiefly neurotropic cataphoretic velocity; the pasteurized milk at the same time yielded streptococci having velocity characteristic of those obtained from normal persons. Both yielded streptococci having fairly normal velocity, with a high incidence of organisms of velocity characteristic

of those found in the presence of the autumnal head cold after the epidemic had subsided, and when head colds had become prevalent in the community (Fig. 2).

CATAPHORETIC VELOCITY OF THE STREPTOCOCCUS

The cataphoretic velocity of the streptococcus was determined with the Northrop-Kunitz Mudd apparatus at about 120 volts, using the zinc electrodes. Inoculations of nasopharyngeal swabbings and of the milk were made directly into previously warmed, tall tubes of glucose-brain broth. The inoculated tubes were incubated at 35° C. for from eighteen to twenty-four hours, when about 2 c.c. of the top layer were poured into clean test tubes. The streptococci were thrown down in the centrifuge at 1,200 revolutions per minute for ten minutes, the supernatant broth was poured off, the tubes were drained thoroughly, and the sedimented bacteria were suspended first in about 2 c.c. of distilled water, and this suspension was then made up to 14 c.c. The final suspension was then poured into the cataphoretic cell and the time required for from ten to twenty diplococci, depending on the evenness of their rate to traverse the unit distance of 50 microns, determined with a stop watch. The effective electric gradient in the cell, under these conditions, was approximately 7.8 volts per centimeter. The cataphoretic velocity in the customary unit was determined according to the formula: $\frac{50 \text{ microns}}{\text{time in seconds} \times 7.8 \text{ volts per centimeter}}$. Cultures containing a preponderance of streptococci with velocities of 3.56, 3.20, or 2.91, and those with velocities of 1.68, 1.6 or 1.52 microns per second, per volt per centimeter, are designed as poliomyelitic or neurotropic, because organisms of these velocities predominate in cases of poliomyelitis and other diseases of the nervous system; those with velocities of 2.29, 2.13 or 2.0 microns per second, per volt per centimeter, are designated as arthrotropic, because streptococci of these velocities occur in great preponderance in cases of arthritis; those with a distribution of velocities of streptococci like those from normal throats of human beings in nonepidemic times, in which there is a moderate preponderance of streptococci having velocities of 2.29, 2.13, or 2.0 microns per second, per volt per centimeter, are designated as normal; those with velocities of *circa* 2.56 microns per second, per volt per centimeter, are designated as rhinotropic, because this is the predominating velocity of streptococci isolated from the nasopharynx in cases of acute rhinitis; those with velocities of *circa* 1.83 microns per second, per volt per centimeter, are designated as pharyngotropic, because this is the predominating velocity of the streptococci isolated from the nasopharynx in cases of pharyngitis or sore throat, and those with velocities of chiefly 1.83, 1.60, 1.42 and 1.28 microns per second, per volt per centimeter, are designated as bronchotropic, because they

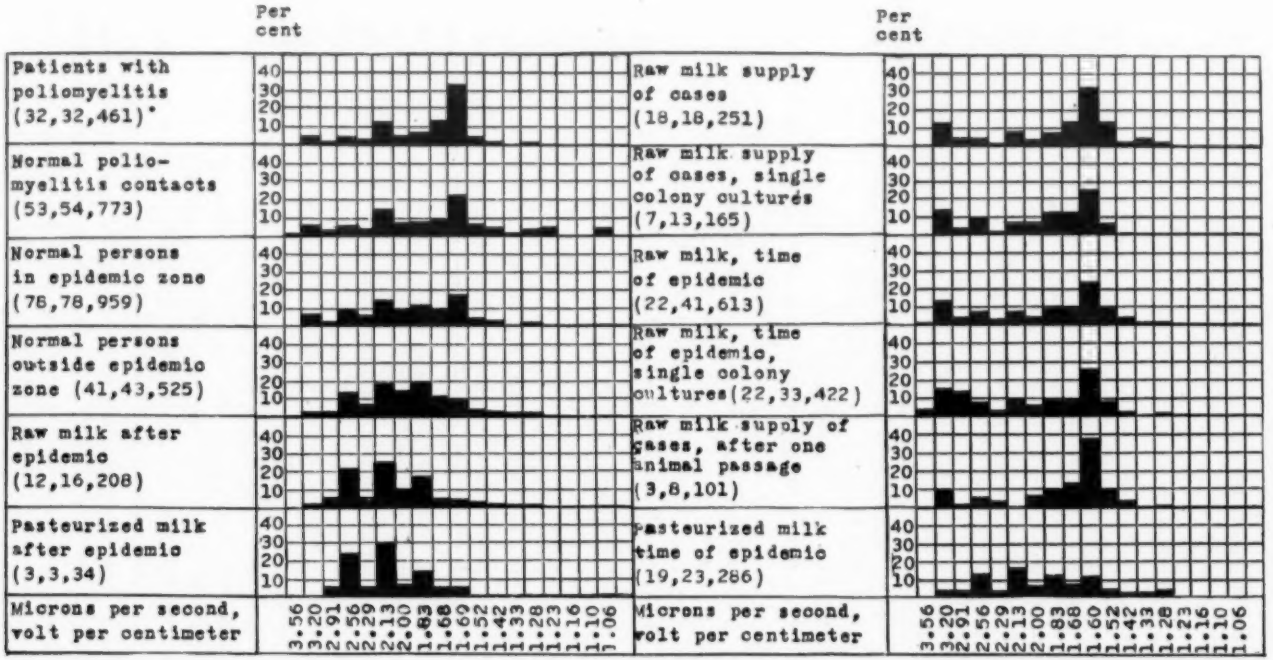
Per cent
Patients with poliomyelitis (32,32,461)*
Normal poliomyelitis contacts (53,54,773)
Normal persons in epidemic zone (78,78,959)
Normal persons outside epidemic zone (41,43,525)
Raw milk after epidemic (12,16,208)
Pasteurized milk after epidemic (3,3,34)
Microns per second, volt per centimeter
3.56 3.20 2.91 2.56 2.29 2.13 2.00 1.83 1.68 1.60 1.52 1.42 1.33 1.28 1.23 1.16 1.10 1.06
Per cent
Raw milk supply of cases (18,18,251)
Raw milk supply of cases, single colony cultures (7,13,165)
Raw milk, time of epidemic (22,41,613)
Raw milk, time of epidemic, single colony cultures (22,33,422)
Raw milk supply of cases, after one animal passage (3,8,101)
Pasteurized milk time of epidemic (19,23,286)
Microns per second, volt per centimeter
3.56 3.20 2.91 2.56 2.29 2.13 2.00 1.83 1.68 1.60 1.52 1.42 1.33 1.28 1.23 1.16 1.10 1.06

Fig. 2.—Graphs of cataphoretic velocity of green-producing streptococci as isolated from nasopharynx and milk in a study of an epidemic of poliomyelitis.

*The figures in parentheses indicate, respectively, the number of strains, cultures, and streptococci timed in each group studied.

are commonly at hand in influenza, affecting chiefly throat and bronchi, and associated with marked constitutional reaction.

The types of cataphoretic velocity displayed by green-producing streptococci from various sources are summarized in Table VI. These sources were as follows: the nasopharynx of patients with poliomyelitis at the Ancker Hospital,* St. Paul, twelve miles distant, where most of the patients from White Bear Lake were taken for treatment; of poliomyelitis contacts; of normal persons within and outside the epidemic zone, and raw and pasteurized milk. The incidence is noteworthy, respectively, of strains and cultures of streptococci having chiefly neurotropic velocity, as isolated in cases of poliomyelitis (88 and 88 per cent), poliomyelitis contacts (60 and 61 per cent), normal persons in the epidemic zone (29 and 29 per cent), in contrast to normal persons outside the epidemic zone (10 and 9 per cent). A similar high incidence of streptococci of neurotropic velocity was found in the strains and in cultures from raw milk during the epidemic (89 and 89, 86 and 76, 77 and 71, 91 and 73, and 100 and 87 per cent respectively). This incidence was in sharp contrast to the comparatively low incidence of streptococci of neurotropic velocity, respectively, in strains and cultures from pasteurized milk during the epidemic (26 and 26 per cent) and in both the raw (0 and 0 per cent) and pasteurized (0 and 0 per cent) milk after the epidemic had subsided. The high incidence of neurotropic velocity, respectively in strains and cultures during the epidemic and the low incidence after the epidemic were found to correspond to a similar high incidence during and a low incidence following the epidemic in the actual number of streptococci timed in the different groups (Table VI), varying from 39 to 71 per cent during the epidemic, and from 17 to 25 per cent from persons outside of the epidemic zone. The incidence of streptococci having neurotropic velocity as isolated from pasteurized milk during the epidemic and from raw and pasteurized milk after the epidemic had subsided was uniformly low. The total percentage distribution of velocities of streptococci timed in each of the different groups is summarized graphically in Fig. 2. The configuration of the graphs representing the velocities of streptococci from the following sources, before and after one animal passage, was strikingly similar: the nasopharynx of patients, the nasopharynx of normal persons who came in contact with patients, the nasopharynx of normal persons in the epidemic zone, the raw milk supply of patients as obtained in the market and at the homes of the patients, and the raw milk supply of homes where no cases occurred at the time of the epidemic. The configuration of the graphs representing the velocity of the streptococci as isolated from the pasteurized milk at the time of the epidemic, from the nasopharynx of normal persons

*We wish to express our appreciation of the kindness of Dr. F. G. Carter, superintendent, and of Dr. W. L. Pratt, house physician, Ancker Hospital, in permitting us to study these cases.

TABLE VI

TYPES OF CATAPHORETIC VELOCITY OF GREEN-PRODUCING STREPTOCOCCI AS ISOLATED FROM NASOPHARYNX AND FROM MILK

SOURCE OF STREPTOCOCCI	STRAINS								CULTURES								STREPTOCOCCI TIMED			
		PER CENT								PER CENT								PER CENT		
	NUMBER	NEUROTROPIC	ARTHROTROPIC	NORMAL	RHINOTROPIC	PHARYNGOTROPIC	BRONCHOTROPIC	OTHER	NUMBER	NEUROTROPIC	ARTHROTROPIC	NORMAL	RHINOTROPIC	PHARYNGOTROPIC	BRONCHOTROPIC	OTHER	NUMBER	NEUROTROPIC	ARTHROTROPIC	OTHER
Patients with poliomyelitis	32	88	6	6	0	0	0	0	32	88	6	6	0	0	0	0	461	57	25	18
Poliomyelitis contacts	53	60	11	13	2	0	13	0	54	61	11	13	2	0	13	0	773	47	27	26
Normal persons in epidemic zone	78	29	13	37	9	7	4	0	78	29	13	37	9	7	4	0	959	39	32	28
Normal persons outside of epidemic zone	41	10	7	44	17	10	12	0	43	9	7	47	16	9	12	0	525	25	38	37
Raw milk supply of cases	18	89	0	11	0	0	0	0	18	89	0	11	0	0	0	0	251	71	13	16
Raw milk supply of cases (single colony cultures)	7	86	0	14	0	0	0	0	13	76	0	8	8	8	0	0	165	64	15	22
Raw milk, time of epidemic	22	77	0	9	9	0	5	0	41	71	0	7	10	2	10	0	613	60	15	25
Raw milk, time of epidemic (single colony cultures)	22	91	0	5	5	0	0	0	33	73	6	9	6	3	3	0	422	64	18	17
Pasteurized milk, time of epidemic	19	26	16	16	21	5	16	0	23	26	13	13	22	9	17	0	286	37	30	32
Raw milk, time of epidemic, after one animal passage	3	100	0	0	0	0	0	0	8	87	0	13	0	0	0	0	101	70	9	21
Raw milk after epidemic	12	0	8	25	33	25	8	0	16	0	13	19	38	25	6	0	208	17	39	44
Pasteurized milk after epidemic	3	0	33	33	33	0	0	0	3	0	33	33	33	0	0	0	34	18	44	38

outside the epidemic zone, and from the raw and pasteurized milk after the epidemic, was also strikingly similar, but very different from that of the aforementioned groups. The high incidence of streptococci having neurotropic velocity, as derived from patients, from persons who came in contact with patients, from the raw milk supply of patients, and from animals that received injections of cultures from raw milk are shown in Table VI and Fig. 2. In contrast, the low incidence of streptococci having neurotropic velocity in pasteurized milk, and a consistent diminution in the incidence of streptococci having neurotropic velocity as derived from throats and from raw milk, as one gets farther and farther away from where cases occurred, and from the time of the epidemic, are also well shown in Table VI and Fig. 2. Also, there was an increased incidence of streptococci having "rhinotropic" velocity (*circa* 2.56 microns per second, per volt per centimeter) in the milk, concomitantly with the prevalence of autumnal head colds in the community after the epidemic of poliomyelitis had subsided. Streptococci having this potential, or velocity, were found not only in bulk samples of milk but also in the milk obtained in a sterile manner directly from cows. Just as the cases were widely scattered, so was the prevalence of the streptococcus in the raw milk from the widely scattered small dairies supplying milk.

One or more samples of each of the eleven brands of raw milk and three of the five brands of pasteurized milk yielded the streptococcus having chiefly poliomyelitic or neurotropic cataphoretic velocity. The brand of raw milk supplied where six cases developed yielded the "poliomyelitis" streptococcus in all of nine samples obtained on four different occasions. Four of these cases occurred about one week before, and two, after our study had begun. This brand of raw milk was supplied to only thirty-eight of the children of school age, of whom six (16 per cent) subsequently had poliomyelitis. All of four samples of one brand of raw milk obtained October 13 and 20, supplied where one of the early cases occurred, yielded the "poliomyelitis" streptococcus, whereas the three samples obtained in November proved free. All of three samples of a brand of raw milk supplied to homes where two cases occurred, obtained October 20, November 4 and 14, the last specimen obtained at the home where one of the cases occurred, yielded streptococci of poliomyelitic potential. Only one sample of raw milk was obtained of the brand supplied to three of the homes in each of which one case occurred, and this one was heavily seeded with streptococci. Both of two samples of this brand of milk obtained, one from the home at about the time a case occurred and one from a previously unopened bottle on the route, about two weeks before the mentioned cases occurred, also yielded the streptococcus. All of the pasteurized samples collected October 13 and 20 proved free from this streptococcus. Of one of the brands of pasteurized milk obtained from previ-

ously unopened bottles each of four samples proved free of the "poliomyelitis" streptococcus, whereas the samples obtained November 14 at the home where a case occurred shortly before, yielded the streptococcus. Similarly, one other pasteurized product proved free of the streptococcus during October but yielded it in both of two samples obtained November 14, one from the home where a case occurred and the other from a previously unopened bottle from the market. All of five samples of a third brand of pasteurized milk supplied in a home where two cases occurred proved free from the streptococcus. One of these cases occurred three weeks before our study was begun. One sample was obtained at the home supplied by this dairy shortly after the second case developed. Altogether, eight different brands of milk supplied to homes where fourteen cases developed revealed the streptococcus at about the time each case occurred. The milk supplied to sixteen patients (to fourteen raw milk, and to two presumably pasteurized milk) was shown to contain the "poliomyelitis" streptococcus. This streptococcus was not found in the milk supplied to three patients, representing two brands of pasteurized milk. Cultures were not made in two cases in which the patients were supplied raw milk from private dairies (Fig. 1).

This streptococcus was found alike in samples of milk obtained from previously opened bottles in households where cases occurred, in those obtained from unopened bottles at the dairies, as well as in bottles obtained in the open market. Accompanied by the official milk inspector we inspected seven dairies supplying raw milk in an attempt to find the source of the streptococcus. No cases of poliomyelitis occurred among members of the families or milkers at these dairies; in this respect this epidemic differs from the milk-borne epidemics described by Aycock.[1, 3] The owners of all assured us that none of the cows had had mastitis, that the milk from all was normal, and that all cows had been tuberculin tested. At one dairy the milk from a recently freshened cow was excluded because it was considered abnormally yellow. Cultures from the milk obtained in a sterile manner from this cow yielded the streptococcus in large numbers. Milking was done chiefly by hand at the different dairies, with usual care, in clean barns. The utensils were apparently properly cleansed and sterilized, and refrigeration was found adequate. The milk from all of the dairies was delivered in bottles sealed with the usual paper disk. Corresponding samples of raw and pasteurized milk were cultured on three occasions. Direct platings of the three raw samples each yielded green-producing streptococci, whereas none of the three that had been pasteurized yielded streptococci on blood-agar plates. Cultures in glucose-brain broth were positive as to streptococci in four of the six samples; namely, the three samples of raw milk and one of the samples of pasteurized milk. The cataphoretic velocity of the streptococci from two of the unpasteurized

samples and from the one pasteurized sample was neurotropic. In the other sample the velocity resembled that of streptococci isolated from the nasopharynx of patients having colds.

ANIMAL EXPERIMENTS WITH THE STREPTOCOCCUS FROM MILK

Twenty rabbits were given intracerebral injections, each with 0.1 or 0.2 c.c., and twenty-four mice were given intraperitoneal injections, each with 2 c.c. of the cultures in glucose-brain broth of the streptococci having poliomyelitic velocity, as isolated from nine brands of milk. Of the rabbits which were given injections, seven remained well, representing three brands of raw milk which were supplied where no cases occurred. In seven rabbits, representing four brands of raw milk, all but one supplied where no cases occurred, symptoms resembling poliomyelitis developed, and then the rabbits recovered. Six rabbits died from flaccid paralysis. Suppurative meningitis was not found in any of these six rabbits, but pipettings from brain and spinal fluid yielded pure, or almost pure, cultures of green-producing streptococci in large numbers on blood-agar plates, and streptococci that grew in short chains in glucose-brain broth; all of these streptococci had chiefly poliomyelitic cataphoretic velocity. The blood of four of the six rabbits that died of flaccid paralysis was sterile, and that of two was lightly seeded with streptococci. Five of the six rabbits that died of flaccid paralysis received cultures from two different samples of raw milk supplied to families in which six cases occurred, and the remaining one rabbit received the streptococcus of poliomyelitic velocity isolated from the chocolate flavored, unpasteurized milk, that was supplied at the Washington School, where three cases occurred. Twelve mice survived, two of which received cultures from the milk supplied where six cases occurred, and the remainder received cultures of streptococci having poliomyelitic velocity from four brands of milk supplied where no cases occurred. Twelve mice, representing three brands of raw milk, died in from one to seven days. Two died of acute colon bacillus infection. Seven of the mice that succumbed to the streptococcus having poliomyelitic velocity, and two that recovered, received cultures representing the first, second and third animal passage of organisms obtained from the brand of milk that yielded the streptococcus in every sample cultured and that was supplied where six of the cases occurred; two mice that died represented two samples of raw milk supplied respectively where two cases and one case occurred, and one mouse that died represented the brand of raw milk supplied where three cases occurred. In none of the mice did serofibrinous peritonitis, pleuritis, or meningitis develop. Streptococci were isolated from pipettings of the brains of all of the ten mice that died of other causes than infection with colon bacilli; in four of these the streptococci were in pure culture and had poliomyelitic velocity. The blood of all the mice

that died from streptococcus infection was either sterile or lightly seeded. The results in these experiments with the streptococcus from the milk were identical to those obtained previously during a study of an institutional outbreak of poliomyelitis also apparently traced to raw milk.[7] The streptococcus from the milk, after the epidemic had subsided, did not cause death of rabbits from paralysis and did not invade the central nervous system following intraperitoneal injection of mice. The results in the following experiment will suffice to illustrate in general the course of events following intracerebral injection of the streptococcus having poliomyelitic velocity from one sample of milk of the brand apparently responsible for six cases of poliomyelitis.

Fig. 3.—Cross-section of cervical portion of spinal cord. Extreme degeneration and lysis of ganglion cells in the anterior horn may be noted (hematoxylin and eosin ×95).

A white rabbit of medium size (1,300 gm.) was given an injection far forward in the right frontal cerebral lobe October 14, 1931, of 0.2 c.c. of the primary culture in glucose-brain broth of the streptococcus having neurotropic velocity.

The following morning the hind extremities of the animal were somewhat tremulous and weak. Its eyes were congested and prominent; there was no paralysis. The animal was given another injection with 0.1 c.c. of the culture in glucose-brain broth, obtained from a single colony on the primary blood-agar plate. The cataphoretic velocity of both cultures was typical of streptococci as isolated from patients with poliomyelitis.

October 16, marked weakness of the hind extremities was noted; the animal was unable to bear its weight on them, and pulled itself along with its front extremities. Respirations were increased. Tremors and excitation had largely disappeared. Following this, typical, slowly ascending, flaccid paralysis supervened, becoming complete, in turn, of hind extremities, muscles of abdomen, back and

chest, fore extremities, neck, and finally of diaphragm. The animal died of respiratory failure eight days after the first injection.

Necropsy revealed nothing of note. The brain and spinal cord were soft, but there were no gross lesions. The meninges were moderately hyperemic. The cerebrospinal fluid was slightly clouded. There was no mark at the point of injection. Cultures from pipettings of the brain yielded a large number of green-producing colonies on blood-agar plates, and a diffuse, pink growth of streptococci that grew in short chains, having chiefly poliomyelitic velocity in glucose-brain broth. The blood proved sterile.

Paraffin sections of the brain and spinal cord, fixed in 10 per cent formalin, revealed circumscribed areas of slight, perivascular infiltration of the meninges by lymphocytes and polymorphonuclear leucocytes, especially in the sulci at the base of the brain, and over the anterior aspect of the spinal cord and of the central canal. There were slight perivascular and parenchymatous infiltration, and localized hemorrhage and edema of the pons and anterior horns of the spinal cord; but the most

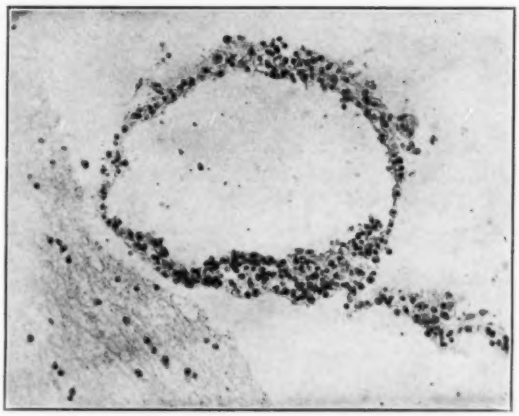

Fig. 4.—Cross-section of a large vein, lower level of pons, of the rabbit cited in the protocol. There are two elliptical areas of infiltration of the wall (hematoxylin and eosin ×100).

striking lesions were edema, necrosis, degeneration, and absence of most of the ganglion cells in the anterior horns of the spinal cord (Fig. 3). A moderate number of gram-positive diplococci of large, moderate, and small size, sometimes in short chains, was found in the regions of perivascular and parenchymatous hemorrhage and infiltration. Several of the larger veins in the meninges, over the anterior aspect of the pons and cervical portion of the spinal cord revealed elliptical areas of infiltration, extending usually only part way around the circumference (Fig. 4). The infiltration was by large and small lymphocytes and polymorphonuclear leucocytes (Fig. 5), but involving locally the entire wall of the vein, especially the intima. Gram-positive diplococci were demonstrated in the adventitia, adjacent to the more normal parts of the wall, and within the areas of infiltration (Fig. 6). Some of these areas resembled closely the lesions of veins described by Wickman,[9] Flexner and Amoss,[2] and more particularly the intravascular lesions of spinal veins found by Saddington[8] in a Macacus cynomolgus monkey, in which poliomyelitis was produced by feeding the virus. A few diplococci were found in the anterior horns, but only after long search, whereas in the normal

structures of brain and spinal cord bacteria could not be demonstrated. Degeneration, with lysis of ganglion cells in the anterior horn, seemed to be due to a toxin, formed partly in situ and partly in more remote structures, such as in perivascular spaces, and having specific affinity for these cells. The motor cells of the cortex and basal ganglia gave evidence of only slight degeneration.

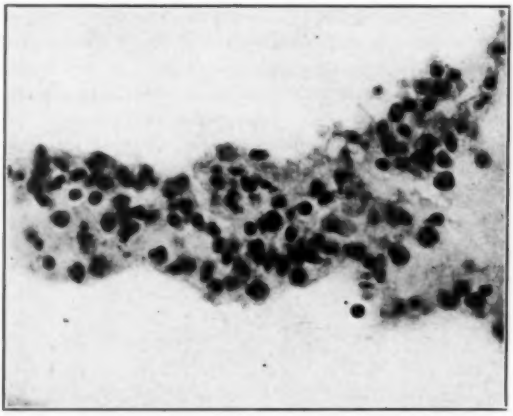

Fig. 5.—The larger area of infiltration of the vein shown in Fig. 4. Large and small lymphocytes are in preponderance over polymorphonuclear leucocytes (hematoxylin and eosin ×810).

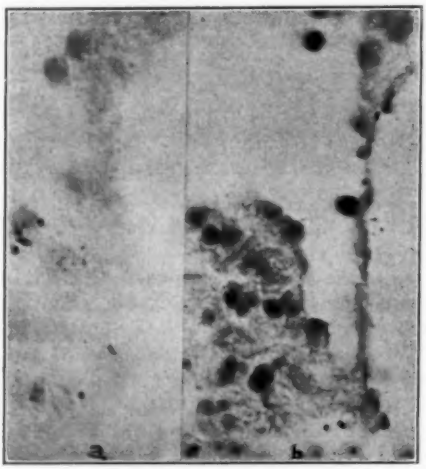

Fig. 6.—Diplococci (a) within endothelial cell and (b) free in the wall of the vein shown in Figs. 4 and 5 (gram stain ×1000).

The cultures in glucose-brain broth which contained a preponderance of streptococci having characteristic cataphoretic velocity as isolated from the nasopharynx of patients, from the milk supply, and from pipettings of the brain and spinal cord of animals that were given in-

jections of streptococci isolated from milk, were used to inoculate large amounts of 0.2 per cent dextrose broth, for studies of cataphoresis, agglutination and precipitin reaction. The twenty-four-hour growth was centrifuged and the sediment suspended in glycerin (2 parts) and 25 per cent sodium chloride solution (1 part) so that each cubic centimeter of this menstruum contained the growth from 75 c.c. of the broth. These dense suspensions were kept in the refrigerator for about seven months. Dilutions were then made for studies of agglutination, precipitin and cataphoretic velocity, with the poliomyelitis antistreptococcus horse serum, and as controls, with an arthritis antistreptococcus horse serum and normal horse serum. The strains from the throats and from the milk, before and after animal passage, reacted essentially alike, and were characteristically positive to agglutination and to precipitin tests in the poliomyelitis antistreptococcus serum, and less so or not at all in the control serums. Thus, each of six cleared extracts of suspensions in sodium chloride solution, of a density equivalent to that of a broth culture, representing six strains of streptococci, each having neurotropic or poliomyelitic cataphoretic velocity as isolated from the nasopharynx of patients with poliomyelitis, and each of four strains isolated from rabbits that succumbed to paralysis, yielded markedly positive precipitin reactions in each of two poliomyelitis antistreptococcus horse serums, and negative or slightly positive reactions in a similarly prepared arthritis antistreptococcus horse serum, and only negative reaction in normal horse serum. Likewise, seven strains of streptococci having characteristic velocity as isolated from raw milk at the time of the epidemic, and each of six strains obtained from milk, as isolated from the brains of paralyzed rabbits, reacted in a manner identical to that of strains obtained from patients. A similar number of strains of streptococci from patients with arthritis, which strains had arthrotropic velocity, reacted markedly with the arthritic antistreptococcus serum and only slightly with the poliomyelitic antistreptococcus serums, and not at all with normal horse serum. Moreover, the poliomyelitic antistreptococcus serum had marked specific cataphoretic velocity-slowing effect and human convalescent poliomyelitis serum had moderate specific cataphoretic velocity-slowing effects, alike over the strains isolated in cases of poliomyelitis of human beings, and over the strains derived from milk.

COMMENT

The epidemic was peculiar in that it occurred late in the autumn, in several explosive outbreaks, during or immediately after spells of warm weather (Fig. 1). It was peculiar also in that an unusual number of multiple cases occurred at about the same time in family groups.

By means of the precipitin reaction between cleared extracts of nasopharyngeal swabbings and the poliomyelitic antistreptococcus se-

rum, the incidence of carriers of the "poliomyelitis" streptococcus was quickly determined and reported to the school and health authorities. By means of a skin test with an appropriate poliomyelitic streptococcus antigen, the presumed susceptibility to poliomyelitis of the entire school population was likewise determined and reported. The children who were both carriers of the streptococcus and positive to the skin test (susceptible) were especially watched. Diagnoses, in consequence, were made early, and serum was then given promptly, and seemingly there was resultant marked reduction in the incidence and degree of paralysis.

The use of raw milk was seemingly responsible for the outbreak. Raw and pasteurized milk were used in the town in about equal amounts. Frank and abortive cases, and the presence of the streptococcus in the throat, were far more common among children who used raw milk than among those who used pasteurized milk. The streptococcus was isolated from all of eight brands of raw milk supplied to families in which fourteen cases occurred. The virulence and incidence of streptococci having poliomyelitic velocity were especially high on isolation from those brands of raw milk that apparently were responsible for the occurrence of the largest number of cases. In an institutional outbreak of poliomyelitis traced to raw milk, reported previously,[7] cases ceased to occur as soon as the use of the contaminated milk was discontinued. Owing to controversy regarding the relative safety of raw or pasteurized milk, the advice given as soon as the streptococcus was found in the milk, namely, that all milk be scalded, boiled or pasteurized, was not generally heeded, and in consequence, cases continued to occur coincidental with, or following, several spells of warm weather.

The occurrence of this streptococcus in several of the samples of pasteurized milk indicates one of three possibilities: (1) the particular samples may not have been properly pasteurized (the temperature and time of exposure necessary to kill this streptococcus are well within the limits used in commercial pasteurization); (2) the streptococcus isolated may have represented contamination after pasteurization; or (3) the samples in which the streptococcus was found may not have been pasteurized, for it was learned that as the milk supply in autumn became scarce, dairymen on the route who delivered both pasteurized and raw milk, according to preference of the family, often procured milk, mostly raw milk, from outside sources to supply their regular customers. This point could not be definitely settled. The finding of the streptococcus in the raw milk of a number of widely separated dairies has, it would seem, great epidemiologic significance, because (1) if a disease presumably due to milk cannot be traced to one milk route, such source is not usually considered worth studying, and because (2) it indicates that the widespread occurrence of this streptococcus among patients was due to reasons other than contact. No cases occurred in

families at the respective dairies; this experience was different from that reported by Aycock[1, 3] following studies of milk-borne epidemics.

It should be emphasized that previous efforts, by standard bacteriologic methods, to isolate the "poliomyelitis" streptococcus from the milk supplies during epidemics of poliomyelitis were unsuccessful. Success in this study, in the institutional outbreak of poliomyelitis,[7] and in isolating the streptococcus uniformly from the milk obtained in a sterile manner from cows where isolated cases occurred on farms during the summer and autumn of 1931, is attributable to cataphoretic studies and to the use of the special medium, glucose-brain broth. This medium, as used, affords a gradient of oxygen tension, and during the short period of incubation is virtually a differential medium for streptococci. Characteristic cataphoretic velocity and virulence of streptococci are maintained in it through successive culture generations, especially if subcultures are made at frequent intervals.

The occurrence of the epidemic after the supply of ice of the town was exhausted, leading to inadvertent, inadequate refrigeration after delivery of the milk, and probable growth of the "poliomyelitis" streptococcus, especially in the raw milk, and the increased incidence of cases on several occasions coincident with the spells of warmer weather, indicate strongly that the epidemic was due to streptococci in raw milk, and that unusually heavy inoculation was necessary to overcome the high general resistance of human beings. The fact that no cases occurred in the families of those who supplied the contaminated milk is in accord with this concept, because refrigeration at the dairies was adequate.

The finding of the streptococcus having neurotropic velocity and virulence in milk should not lead to a general diminution in the consumption of milk, this most wholesome of foods, but should lead to a greater safeguarding against organisms potentially virulent to man, so commonly found in milk. Since streptococci having neurotropic velocity and virulence have been isolated from the milk obtained in a sterile manner from healthy cows, having no sign of disease of the udder, and no evidence of inflammatory reaction such as increased numbers of leucocytes in the milk, and since milk is a good culture medium, ordinary methods, such as inspection of herds, bacterial counts, and certification, as generally carried out, and as was done in this outbreak, will not suffice. Proper pasteurization and refrigeration seem necessary and most practical unless some other, simpler and more effective method can be found.

There are a number of facts which indicate that the neurotropic velocity of the streptococci in the raw milk at the time of the epidemic, represents a temporary, acquired property of green-producing streptococci commonly present in the milk of healthy cows, and that the pres-

ence of the streptococcus was not due to contamination from human beings, as is usually assumed and as, indeed, is most likely in milk-borne epidemics of this and other streptococcal diseases. The presence of green-producing streptococci was widespread in the raw milk from the different, widely separated dairies, with no history of exposure to cases of poliomyelitis. The cataphoretic velocity of the streptococci isolated at the time of the epidemic was neurotropic; after the epidemic, it was similar to that of streptococci from normal throats in nonepidemic times. The neurotropic properties of the streptococcus disappeared on artificial cultivation as they acquired a different, more normal type of velocity; just as did those of streptococci isolated from bulk milk, from milk obtained in a sterile manner directly from cows, and from the nasopharynx of human beings after the epidemic had disappeared (Table VI, Fig. 2). Streptococci isolated from milk as the epidemic was subsiding and that had a normal type of cataphoretic velocity, and those isolated during the epidemic which had neurotropic cataphoretic velocity on isolation, and that had acquired a normal type of velocity, acquired characteristic neurotropic velocity and virulence following injection into rabbits and mice, and in turn both groups of strains again acquired the normal type of velocity on further cultivation on artificial mediums. In an institutional epidemic of poliomyelitis traced to raw milk,[7] and in the isolated cases on farms during the summer and autumn of 1931, similar shiftings in the cataphoretic velocities of the streptococci occurred simultaneously in the throats of patients and contacts, and in the milk obtained from cows in a sterile manner. The type of velocity of the streptococci prevalent at the time when poliomyelitis occurred became like that of streptococci as isolated from throats of normal persons in nonepidemic times, in the milk of cows obtained in a sterile manner, in the bulk raw milk, in the throats of contacts, and in the throats of patients while under quarantine. Regardless as to whether the "poliomyelitis" streptococcus found in the milk represented contamination from human sources, or streptococci commonly present in the milk in the udders of cows, which had acquired poliomyelitic properties, the need for pasteurization and adequate refrigeration is apparent. A heavy contamination, veritable cultures of the streptococci, in the milk seemed necessary to infect. The possibility that the streptococcus is an index of the presence of the virus, or that some other relationship exists between the streptococcus and virus, must also be considered.

The streptococcus isolated from the raw milk at the time of the epidemic was identical with that isolated in cases of poliomyelitis in human beings and monkeys, in its peculiar virulence, in its power to cause flaccid paralysis, especially in rabbits, in its thermal death point, in its morphology and cultural characteristics, in its immunologic properties, and in its cataphoretic velocity. Moreover, the serum from persons and monkeys who had recovered from poliomyelitis had specific velocity-

reducing effect on the streptococcus isolated from raw milk, similar to the effect which it has on the streptococcus isolated in cases of poliomyelitis.

SUMMARY

The results of an epidemiologic and experimental study of an epidemic of poliomyelitis, apparently due to raw milk, are reported.

The etiologic importance in acute epidemic poliomyelitis of streptococci having characteristic cataphoretic velocity and virulence and specific antigenic properties, that have been described repeatedly previously, is again emphasized by the results of this study.

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CHRONIC SIALODOCHO-PAROTITIS WITH RECURRENT SUBACUTE EXACERBATIONS

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THE condition under discussion is characterized by recurrent, rapidly appearing, occasionally painful swellings of one or both parotid glands, most often without any apparent cause, and associated frequently with a discharge of pus or cloudy saliva from Stenson's duct. Since this somewhat unusual condition is only rarely mentioned and briefly discussed in the English and American literature, we thought it of interest to report a typical case occurring in a child.

The following causes of chronic enlargement of the parotid glands are found in the literature: Tumors[1] (benign and malignant); foreign bodies in the duct including salivary calculi[2]; the specific infections (tuberculosis,[3] actinomycosis and syphilis[4]); Mikulicz's disease; certain types of obesity[5]; hyperplasia of the gland tissue occurring in disturbances or diseases of the genital organs and of some of the endocrine glands[3, 4, 6, 7, 8]; leucemia; gout; hereditary familial chronic swelling of the parotid glands[9]; "salivary retention" described by Kien[10] in two newborn infants; malaria (Gros quoted by Küttner[1]); following mumps[11]; and drug intoxications (lead, iodine, copper, and opium[3]); and chronic sialodocho-parotitis with recurrent subacute exacerbations.

CLINICAL ASPECTS

The swelling of the parotid gland or glands in this condition, as previously stated, has a tendency to recur and may be slightly painful. It may last from several hours to several days or weeks, the gland remaining moderately enlarged between exacerbations. In some instances, as in the cases of Keith[12] and Hewlett[13] the swelling appears in the morning and disappears after breakfast. The last named author explains this on the basis of a decreased salivary secretion during the night with a consequent tendency to duct obstruction. Salivary secretion is usually not affected; although the patient of Pyrah and Allison[14] complained of excessive discharge of pus and saliva from the affected gland, and one of LeClerc's[15] cases had a constant drooling of saliva

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from the involved side. There is as a rule no febrile reaction, and the general health is apparently unaffected. The patient may learn to obtain relief by pressure over the gland with the resultant discharge of pus or saliva, or as in the condition of "fibrinous sialodochitis," by removal of a plug of fibrin from the duct. The latter condition was first described by Kussmaul[16] and has been observed in children by Noeggerath[17] and Johnson.[18]

There is usually no apparent predisposing cause, although cases following tonsillectomy, tonsillitis, and tooth extraction have been reported,[12, 19] and Von Reuss[3] observed one case in an eight-year-old girl who had exacerbations with recurrent upper respiratory infections.

On examination during the recurrences, one finds an enlarged, firm, occasionally nodular gland, which may be slightly tender to pressure. The overlying skin generally shows no changes, but at times it has been observed to be red, and in one case, reported by Johnson,[18] it became fluctuant. Incision revealed no pus, however. The duct is not palpable unless it is stenosed[2] or there has been a long standing fibrinous sialodochitis.[20] The duct opening and papilla may or may not be red. Pressure on the gland may cause the appearance of pus, a cylindrical mass of fibrin and pus, cloudy saliva or unaltered saliva.

Suppuration is very rare but may occur.[19, 21] At times, air has been found in the duct or in the gland; C. Walther[22] thought this complication is due to a mechanical forcing of air into the dilated inflamed duct during mastication.

Table I summarizes the cases in children reported in the literature and includes our case. Analysis shows that 13 of the 18 cases had unilateral involvement and only 5 were bilateral. The ages of the patients varied from one month to sixteen years, and the duration of the disease, when the cases were seen by the observers, from one month to six and one-half years. As far as sex is concerned, the cases are evenly divided. Lipiodol injections into Stenson's duct were done by Keith,[12] and Pyrah and Allison,[14] besides ourselves. The roentgenograms thus obtained by the first named authors showed a generalized dilatation of the smaller ducts and of the main duct, while the lipiodol visualization in our case revealed a dilatation of the smaller ducts only (Fig. 1). This is possibly due to the fact that our case is not of as long duration as those of the other authors. The latter have also injected some normal parotids and were able to visualize in the normal glands only the main duct and a few of the smaller ducts at the hilum of the gland. The narrowing of a portion of the duct shadow which appears in the roentgenogram of our case, is also present in the normal duct shadows obtained by Keith[12] and Pyrah and Allison,[14] and, according to Pyrah and Allison, this attenuation is present where the duct crosses in front of the masseter muscle.

TABLE I

CASES OF CHRONIC SIALODOCHO-PAROTITIS WITH RECURRENT SUBACUTE EXACERBATIONS IN CHILDREN, OCCURRING IN THE LITERATURE

AUTHOR	YEAR OF REPORT	AGE	SEX	UNILATERAL	BILATERAL	DURATION WHEN SEEN BY AUTHOR	THERAPY	REMARKS
Johnson, R.	1896	13	Male	+		1 month	Not mentioned.	Fibrinous sialodochitis; became fluctuant. Incision showed no pus.
Johnson, R.	1896	11	Male	+		1 month	Hot douches.	
Johnson, R.	1896	15 mo.	?	+		5 weeks	Not mentioned.	
Tietze, A.	1905	4 mo.	Male	+		6 weeks	Removal of gland.	Microscopic examination: Chronic inflammatory picture with ameba-like bodies in smaller ducts.
Noeggerath, C. T.	1909	13	Male	+		¾ year	Massage, good general care, diet, iodides as for a bronchitis.	Fibrinous sialodochitis. Smear of pus showed polys—bacteria—and epithelial cells. Improvement noted.
Von Reuss	1909	8	Female		+	6 years	X-ray.	Pain with exacerbations. Improvement.
Von Reuss	1909	4	Male	+		6½ years	None mentioned.	Apparently no recurrences. Only a constant swelling present.
Von Reuss	1909	8	Female		+	"Several years"	None mentioned.	Attacks of abdominal colic, occasionally coincidental with parotid swelling.
Thaysen	1910	3	Female	+		6 months	Removal of gland.	Microscopic examination: Chronic inflammatory process with replacement of gland tissue by connective tissue.

TABLE I—CONT'D

AUTHOR	YEAR OF REPORT	AGE	SEX	UNILATERAL	BILATERAL	DURATION WHEN SEEN BY AUTHOR	THERAPY	REMARKS
Neuhof	1916	4	Male	+		5 months	Repeated dilatation and slitting of stenotic portion of duct.	Stenosis of duct. Duct palpable. Improvement.
Neuhof	1916	8	Female	+		4 months	Removal of stenotic portion of duct.	Stenosis of duct. Recovery.
Blair & Padgett	1923	6	Male	+		2 years	Slit duct. Massage.	Improvement.
Blair & Padgett	1923	10	Female	+		3-4 years	Incision.	Small abscess cavity found.
Wolff, S.	1926	1 mo.	?		+	?	Not mentioned.	Recurrent rapid swellings associated with enterospasm.
Keith, H. M.	1928	13	Female	+		6 years	Dilatation of duct.	Lipiodol injection shows duct dilatation especially involving smaller ducts at hilum. Followed tonsillectomy. Culture of pus: Strep. viridans.
Steinitz, F.	1929	4	Female		+	2 years	None mentioned.	Rapidly occurring swelling, once under observer's eyes. Pilocarpine injection caused swelling. Clear saliva on pressure. Author thinks condition one of duct spasm.
Pyrah & Allison	1931	16	Female		+	4 years	Massage. Dilatation of duct. General care. Iodides.	Excessive discharge of saliva and pus with exacerbations. Culture: Staph. Lipiodol injection. Same findings as in Keith's case.
Londe & Pelz	1932	10	Male	+		2½ years	Milk injections.	Lipiodol. Dilatation of smaller ducts. Culture: *B. fecalis alcaligenes.*

PATHOLOGY AND ETIOLOGY

The pathology has been described by Tietze[23] who obtained a six weeks' enlarged parotid from a four-month-old infant, and by Thaysen[24] who obtained his specimen from a three-year-old child whose right parotid had been swollen for six months. Both authors described essentially the same picture of chronic and subacute inflammation; i.e., a marked increase of interlobular connective tissue, with more or less of a fibrous tissue invasion of the lobules. There was also present a marked round cell infiltration, with occasional lymphoblasts, plasma cells, leucocytes and giant cells. In places, the parenchyma was replaced by the inflammatory tissue. Tietze, in addition, found ameba-like protozoa in the smaller ducts, but since these organisms are found normally in the mouth, the author felt them to be secondary invaders. The main ducts are not described, but Pyrah and Allison,[14] who excised a chronically inflamed sublingual gland, found the duct of this gland to be dilated and fibrotic, the wall being almost cartilaginous in consistency.

As far as the etiology of this condition is concerned, it is generally accepted that the infection begins in the duct, this being followed by recurrent obstructions with fibrin, mucus, pus, etc. It seems to us that the general involvement of the smaller ducts as visualized by lipiodol injection is confirmatory evidence of this theory.

Although various organisms[12, 14, 19] have been found in the discharge obtained from the duct, it should be remembered, as Kroiss[25] has pointed out, that bacteria have been found in the excretory ducts normally. However, the smaller ducts and gland tissue have been found to be sterile. Claisse and Dupré (quoted by Kroiss[25]) could not produce inflammation in the salivary glands of dogs by the injection of bacteria and irritants, or foreign bodies into the ducts. Since experimental obstruction of the duct of a salivary gland merely causes an atrophy of the gland tissue and no increase in connective tissue as occurs in chronic inflammation, Kroiss concluded that the changes occurring in inflammatory conditions of salivary glands are due to infection. In view of the fact that the gland tissue and smaller ducts are normally resistant to mouth organisms by a washing out of the bacteria by means of the saliva or for other reasons, the same author felt that there must probably be some predisposing factor which disturbs the secretion of the glands before infection can occur. However, the possibility that inflammatory change might be caused by partial obstruction of the duct was not considered by Kroiss.

Such conditions as surgical operations, generalized infections, infection of the mucous membranes of the mouth, and starvation as in treatment of gastric ulcer with inhibition of the salivary secretion,

have been observed to cause infection of the parotids. Such predisposing factors are not so apparent at first glance in the condition reported in this paper.

Reflex spasm of the ducts and allergic swelling of the glands have been advanced by various authors as the predisposing cause of this disease. Thus LeClerc[15] suggests that the condition may be due to a "dystrophy of the duct." Wolff[26] described a case of recurrent swelling of both parotids in an infant with enterospasm and felt that he was dealing with reflex spasms of the duct. The case of Von Reuss[3] (see Table I) with abdominal colic, at times coincident with the glandular swelling, may fall into this category. Steinitz[27] also had a case in which he thought spasms of the duct occurred. Vogeler,[20] finding an 11 per cent eosinophilia in his case, likened the condition to an asthma, while Burton-Fanning[28] described the interesting case of a man sixty-two years old, who had been having recurrent attacks of sneezing, dryness of the mouth and swelling of the parotid glands. Injection of adrenalin caused a more rapid recession of the swellings than had ever before occurred.

In view of the above observations, it is possible that due to some general or local reflex disturbance of the glandular secretion or of the innervation of the duct, a predisposition to infection is established. Kroiss[25] felt that where infection occurs a chronic condition at times results because the glands are still partly resistant to the invading organisms.

REPORT OF CASE

M. G., a male, aged ten years, was admitted to the hospital, August 11, 1932.

Present Illness.—Two and a half years before his admission, the patient developed overnight a painful swelling of the left side of the face. There was some fever, and the patient's mother thought he had mumps, although to her knowledge there had been no exposure and no mumps in the neighborhood. The swelling subsided in several days, but since then had recurred every three to four months, appearing most often in the morning and being painful with mastication. These recurrences lasted several days to a week and were not associated with any disturbances in salivary secretion. The last swelling had lasted for three weeks.

In addition, for the past three years the patient had been having indefinite attacks of nonradiating sticking pain in the left upper quadrant. These attacks were occasionally associated with nausea, but were neither related to meals nor coincident with the parotid gland swellings.

Past history and *family history* were irrelevant.

Physical examination (positive findings only).—Some caries of the teeth. A visible, firm, nontender swelling apparently involving the entire left parotid gland. The overlying skin showed no changes, nor was it unusually warm to touch. Neither Stenson's duct nor any foreign body in it was palpable. The mouth of the duct was reddened and the left papilla was more prominent than the right. Firm pressure over the gland caused a thin purulent secretion to appear at the mouth of the duct. The anterior and posterior cervical glands were moderately enlarged.

Laboratory findings.—Blood and urine normal. Mantoux 0.1 mg. negative. Kahn negative. Smears of pus from Stenson's duct: no eosinophiles; some diplococci; no acid fast organisms.

Culture of pus from duct: *B. fecalis alcaligenes.*

Roentgenogram of gland: Negative for duct stone.

Course in hospital.—After several days of observation, during which time there was no fever, the patient was given a course of three intramuscular sterile milk injections at the suggestion of Dr. Zentay. Following the last injection of 5 c.c. of milk, the patient's temperature rose to 103°, and the following day the gland was smaller and slightly softer. The swelling then rapidly subsided, but not completely.

Probing of Stenson's duct revealed no obstruction or foreign body. Following this, one of us (M. D. P.) injected about 1.5 c.c. of lipiodol into the duct.

Fig. 1

The roentgenogram (Fig. 1) revealed numerous small dilatations of probably the secretory ducts. The main duct did not appear to be dilated. Following the lipiodol injection there was a slight increase in the degree of swelling; this subsided rapidly, however, and the patient was discharged September 3, 1932.

Course since discharge.—There have been two recurrences since the patient left the hospital, each time the swelling appearing overnight and subsiding within two hours. There have also been several attacks of abdominal pain, one attack appearing a day before the last swelling of the gland. Gastrointestinal series and flat kidney roentgenograms for stone have been negative. The blood diastase and sugar tolerance tests have been normal.

Between recurrences, the left parotid remains palpably enlarged and the duct opening remains slightly reddened. Pressure over the gland causes a discharge of clear saliva. Culture of this saliva and of the saliva from the opposite gland has yielded gram positive and gram negative cocci.

COMMENT

The above reported case is typical of the condition described. The complicating abdominal pain has been of some interest. Whether we are here dealing with some abdominal condition which has reflexly affected the parotid gland is difficult to say. One would, in such case, expect a bilateral swelling. The sugar tolerance test and the blood diastase[29, 30] determination were performed with the thought of possibly establishing the presence of some associated pancreatic disease. As to whether the milk injections had anything to do with the regression of the swelling, while the boy was in the hospital, is of course difficult to say, since previous spontaneous improvements had always occurred.

THERAPY

(1) Nonsurgical. Dilatation of the duct and massage of the gland; general care; eradication of foci of infection, such as infected teeth; iodides; injection of mild antiseptics or irritants (as lipiodol, mercurochrome, etc.) into the duct; x-ray; adrenalin; and foreign protein therapy.

(2) Surgical. (a) Slitting of the duct[19]; (b) slitting or removal of stenotic portion of duct[2]; (c) extirpation of the auriculotemporal nerve with resultant atrophy of the gland;[15] (d) removal of the gland.[23, 24]

SUMMARY

The condition of chronic sialodocho-parotitis with recurrent subacute exacerbations is defined and described.

A typical case, in which the ducts of the involved parotid gland were visualized by means of lipiodol, is reported (Fig. 1).

Some improvement was apparently obtained following intramuscular milk injections.

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University Club Building.
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SPONTANEOUS NONTUBERCULOUS PYOPNEUMOTHORAX AS A COMPLICATION OF PNEUMONIA IN CHILDREN

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SPONTANEOUS pneumothorax is considered by some as that form of pneumothorax due to any internal cause, while others would limit it to that type in which no demonstrable etiologic factor can be found. This latter form is also known as idiopathic pneumothorax.

In this communication we are concerned with the spontaneous nontuberculous type occurring during the course of pneumonia. Johnson[1] and Stoloff[2] have written comprehensive papers on this subject. The latter in 1928 collected 81 instances of spontaneous pneumothorax in children which had been reported since 1844 and contributed three cases complicating pneumonia. Of 59 cases which permitted detail study, 35.7 per cent occurred during the course of pneumonia. Since then additional reports have been made by Rajam[3] and Jones.[4] This low incidence justifies the reporting of four cases of spontaneous pyopneumothorax occurring in 42 consecutive cases of pneumonia.

CASE REPORTS

CASE 1.—S. W., three and one-half years of age, was admitted to the hospital on March 31, 1930, with a six-day history of pneumonia and on examination presented the following picture. A well-developed boy who was dyspneic, cyanotic, profoundly toxic and delirious. Bronchial breathing and dullness were obtained over the entire right lung. The heart action was rapid but regular with poor myocardial tone; the abdomen was distended. The following day convulsions developed involving the left side of the face and upper and lower extremities. Pulmonary edema developed that evening. The temperature fluctuated between 103° and 102° Fahrenheit, respirations between 60 and 50 and pulse 140 to 120. Forced stimulation, oxygen, pituitrin for the distention and lumbar puncture were necessary. He rallied after a few days, but on the tenth day after admission he was suddenly seized with an attack of cyanosis, marked respiratory embarrassment and severe pain in the right side of the chest.

Examination at this time revealed hyperresonance anteriorly, disappearance of tactile fremitus, diminution of breath and voice sounds at the base and lower half of the right side posteriorly, and amphoric breathing in the axilla. Succussion splash was elicited, but the coin test and metallic tinkle were not. The diagnosis of pyopneumothorax was confirmed by the following x-ray findings: "There is an irregular pneumothorax cavity involving practically the entire right side with numerous adhesive bands traversing the cavity in such manner as to prevent com-

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plete pulmonary collapse. A free fluid level is present, the upper level of which reaches the seventh rib posteriorly. The mediastinum is displaced to the left."

An extension of the process was evidenced in a few days by a perceptible lagging of the respiratory excursions on the right side, bulging of the intercostal spaces and a resultant prominence of that side and by the presence of metallic tinkle, coin test, tympany and orthopnea.

The pus was removed by repeated aspirations until the third week when a closed thoracotomy was performed. This was followed by the development of an almost complete pneumothorax which was partly absorbed by the time of discharge. An x-ray film taken at this latter time (May 31, 1930) showed a partial pneumothorax, with the right lung adherent at both the upper and lower parts but no free fluid. There was a marked retraction of the right side of the chest with a compensatory curvature of the dorsal curve in the opposite direction. This scoliosis was observed clinically in the fourth week.

Fig. 1

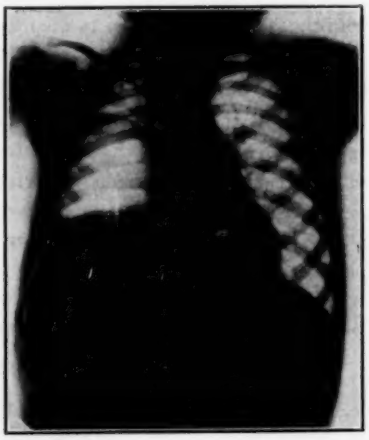

Fig. 2

Fig. 1.—Case 1. April 10, 1930. Erect plate. There is an irregular pneumothorax cavity involving practically the entire right chest. There are numerous adhesive bands traversing the pneumothorax cavity in such a manner as to prevent complete pulmonary collapse. A free fluid level is present, the upper level of which reaches the seventh rib posteriorly. The mediastinum is displaced to the left.

Fig. 2.—Case 1. December 29, 1930. Recurrent pyopneumothorax. Erect plate. There is an irregular pneumothorax cavity involving the greater portion of the right chest with a free fluid level at the level of the ninth rib posteriorly. There are numerous adhesive bands traversing the pneumothorax cavity and permitting only partial collapse of the lung.

The Mantoux test was negative in dilutions of 1:10,000 and 1:1,000. Pneumococcus type IV was isolated from the pleural fluid. The red blood cells ranged from 2,200,000 on admission to 4,200,000 on discharge. The leucocytes ranged from 48,000 to 15,000; there was a decided "shift to the left" by the Schilling method throughout the course. The hemoglobin fluctuated from 60 to 70 per cent (Sahli). A toxic nephritis was present during the entire stay in the hospital.

Second Admission: The child was readmitted on Dec. 27, 1930, with an interim history of a protracted convalescence and the persistence of a nonproductive cough and right sided pain. On December 20, he expectorated a greenish four-smelling mucopurulent material. The next day he had a chill, temperature of 105 degrees

Fahrenheit, an exaggeration of the pain in the right side and respiratory embarrassment which continued until the day of admission.

On examination he presented marked pallor, dyspnea, dilatation of the alae nasi and the following signs in the right chest: The right side of the chest was retracted and showed decreased respiratory excursions. Anteriorly there was dullness to flatness in the upper one-third; posteriorly there were dullness in the middle third and flatness in the base and lower one-third. Amphoric breathing and increased vocal fremitus were found in the upper one-third anteriorly. Posteriorly there was found decreased vocal fremitus in the lower two-thirds, diminished breath sounds over the middle third and absent breath sounds in the lower third and base. Succussion splash and the coin test were elicited. The heart was not displaced; the sounds were regular and of good quality. The next day hyperresonance was noted anteriorly.

A diagnosis of recurrent pyopneumothorax was confirmed by the following x-ray findings: "There is an irregular pneumothorax cavity involving the greater portion

Fig. 3.—Case 1. April 8, 1932. Recurrent pyopneumothorax. Erect plate. There is a localized pneumothorax cavity occupying the lower lateral half of the right chest. The lung is adherent both to the right diaphragmatic leaflet and to the right chest wall. There is a diffuse shading over the upper half of the right chest.

of the right chest with a free fluid level at the level of the ninth rib posteriorly. There are numerous adhesive bands traversing the pneumothoracic cavity and permitting only partial collapse of the lung."

A closed thoracotomy was performed one week later and subaqueous drainage established. A transfusion of 200 c.c. whole blood (Type 3) was given. The boy was discharged after a stormy course on Feb. 6, 1931. An x-ray film taken three days previously showed two areas of pneumothorax with pleuropulmonic adhesions at the right base and collapse of the lower half of the lung.

Streptococcus hemolyticus was isolated from the pleural fluid. The Mantoux test in 1:1000 dilution was again negative. The blood picture was similar to that on the first stay in the hospital.

Third Admission: The boy had been in a fair state of health except for a chronic nonproductive cough and persistent pain in the right side of the chest. Three weeks prior to admission, he had developed measles; for the ten days immediately preceding his readmission, his temperature ranged from 104° to 103° F.

Examination at the hospital on March 21, 1932, revealed moist râles over the right upper and middle lobes and the left lower part of the chest, decreased breath sounds and dullness to flatness at the right base and over the site of the previous operations, but there was no shifting dullness or flatness. The spleen was palpable and the liver was felt just below the right costal margin. X-ray examination on the day of admission revealed no marked material change from the condition present on his second discharge from the hospital. There was a localized pneumothorax cavity occupying the lower lateral half of the right chest with a diffuse shading over the upper half. The lung was adherent both to the right diaphragmatic leaflet and to the right chest wall.

Empyema developed within a few days and thick yellow pus was aspirated. Thoracotomy was decided upon and the boy was removed to the operating room, but before any surgical measures could be instituted, he was seized with a sudden, severe coughing spell which resulted in the expectoration of a large amount of pus. It was deemed best by the surgeon to dispense with any further surgical inter-

Fig. 4.—Case 2. April 18, 1930. Prone plate. There is evidence of practically a complete pneumothorax cavity occupying the left chest. The density on this side is increased owing to the presence of fluid in the most dependent portion. (A free fluid level is not seen because of prone position. In this position the fluid gravitates to the posterior chest and accounts for the increased density.) The left chest is hyperexpanded. The left diaphragm is depressed. The mediastinum is displaced moderately to the opposite side.

vention as there had evidently developed a fistulous opening between the pleural cavity and the bronchi. Pneumococcus type IV was isolated from the pus. There was also a discharge from the old scar. The child was discharged on April 20, 1932. An x-ray film taken a few days previously showed a retraction of the right lung, a slight haze in the immediate costophrenic angle and a localized pneumothorax.

Case 2.—H., sixteen months of age, was admitted on April 8, 1930, with a five-day history of left lobar pneumonia and suppurative otitis media. He was acutely ill, dyspneic and slightly cyanotic. Bronchial breathing and dullness were elicited over the left apex and upper third anteriorly and over the entire left side of the chest posteriorly. There was no cardiac displacement at this time. The temperature was 102° Fahrenheit, respirations 60 and pulse 120. Red blood cells were 3,900,000; hemoglobin, 64 per cent; white blood cells, 39,800; polymorpho-

nuclears, 84 per cent; lymphocytes, 12 per cent and monocytes, 4 per cent. The Mantoux test was negative in 1:1,000 dilution.

His condition remained critical for the next six days and he had a temperature of 104°. A change was noted in the physical signs in the left lower lobe from dullness to flatness and from bronchial breathing to a diminution and then to a complete absence of breath sounds. Cardiac displacement to the right was noted at the end of the first week.

The first signs of a complicating pneumothorax were a change to hyperresonance and amphoric breathing over the affected area. Examination by both the fluoroscope and film in the prone position showed a pyopneumothorax with the following details: "There is evidence of practically a complete pneumothorax cavity occupying the left chest. The density on this side is increased owing to the presence of fluid in the most dependent part. The left chest is hyperexpanded. The left diaphragm is depressed. The mediastinum is moderately displaced to the opposite side."

FIG. 5.—Case 3. March 18, 1930. There is evidence of practically a complete pneumothorax cavity occupying the left chest. The density on this side is increased owing to the presence of fluid in the most dependent part. The left chest is hyperexpanded. The left diaphragm is depressed. The mediastinum is moderately displaced to the opposite side.

Surgical intervention was not resorted to because of the continued improvement. Complete absorption of the pleural fluid had taken place by the time of discharge (April 30) but the pneumothorax was still present. A follow-up taken May 14, 1930, showed that the pneumothorax still persisted in the lower peripheral half of the left thorax. The left lung had expanded to near the periphery and the pleura was somewhat thickened but there was no evidence of fluid. There was no cardiac displacement. The boy was readmitted on Feb. 2, 1931, for an abscess of the left chest wall in the midaxillary line of ten days' duration. It responded to incision and drainage. There was no pneumothorax present at this time.

CASE 3.—B., seven years of age, was admitted on March 16, 1930, with a ten-day history of right lobar pneumonia. Admission examination revealed lagging of the right side of the chest, bronchial breathing, pleuritic friction rub on deep inspiration, scattered coarse râles and dullness merging into flatness over the lower third of the right lung. He appeared acutely ill and had a temperature of 103° Fahrenheit, respiration of 45 and pulse 110. A diagnosis was made of unresolved lobar pneumonia with pleuritic effusion.

X-ray examination the same day revealed the following: "There is a circular area at the right base and greater translucency which may be a localized pneumothorax. There is no evidence of free fluid in either thoracic cavity, although a small amount at the right base cannot be ruled out."

There was an increase in the severity of the condition within the next week as evidenced by a diminution of both breath and voice sounds, disappearance of the râles, increase in the areas and intensity of flatness and dullness and the appearance of a small zone of hyperresonance tending toward tympany at the base. X-ray examination on March 21 showed an opacity at the base of the right lung, obliteration of the right costophrenic angle and an irregular pneumothoracic cavity involving the lower half of the right chest. There was also adhesive pleurisy and thickening of the horizontal interlobar fissure.

At no time were any other signs of pyopneumothorax present such as amphoric breathing, succussion splash, metallic tinkle or coin test. There never was any

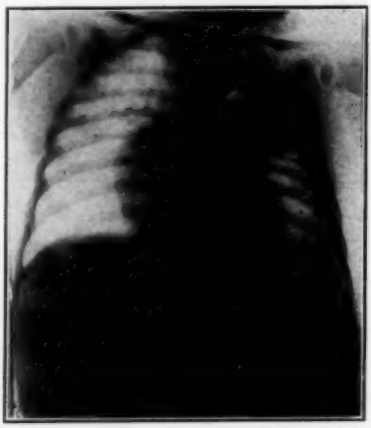

Fig. 6.—Case 4. April 25, 1930. Prone plate. There is a pneumothorax cavity in the left chest extending practically from apex to base. There is a thickened banded shadow along the periphery of the left chest. The left diaphragmatic leaflet is not visible. The mediastinum is displaced to the right. There is retraction of the left chest with narrowing of the interspaces.

cardiac displacement. The child complained of a severe pain in the right side for the first few days. The hyperresonance disappeared within a few days.

Several aspirations of the chest resulted in the expulsion of air without pus. There was no further surgical intervention except for a transfusion of 300 c.c. of whole blood (Type 2). On discharge on April 13, the child presented dullness and slightly decreased breath sounds over the right base. X-ray films taken at this time showed a resolving pneumothorax.

The Mantoux test in dilution of 1:1000 was negative. The blood picture on admission was: red blood cells, 3,840,000; hemoglobin, 65 per cent; white blood cells, 22,800; stab cells, 3; segmented cells, 74; large lymphocytes, 11, and small lymphocytes, 11.

CASE 4.—L. F., one year of age, gave a history of irregular fever since an attack of lobar pneumonia two weeks previously. Admission examination on April 19, 1930, showed a well-developed infant who did not appear acutely ill. The temperature was 100.6° Fahrenheit, respiration 50 and pulse 120. There were

diminished tactile fremitus, diminished breath and vocal sounds and dullness to flatness over most of the left side of the chest. The heart was slightly displaced to the right.

Blood findings were: red blood cells, 2,900,000; hemoglobin, 54 per cent; white blood cells, 13,200; polymorphonuclears, 67 per cent; lymphocytes, 30 per cent; and monocytes, 3 per cent. A diagnosis was made of unresolved pneumonia and empyema.

Routine fluoroscopic and x-ray examination the next day revealed a pyopneumothorax with these details on a prone plate: "There is a pneumothoracic cavity in the left chest extending practically from the apex to the base. There is a banded shadow along the periphery of the left chest. The left diaphragmatic leaflet is not visible. The mediastinum is displaced to the right. There is retraction of the left chest with narrowing of the interspaces." A free fluid level was visualized in the erect position.

Repeated examinations by several members of the pediatric staff in both the erect and the prone positions failed to elicit any of the accepted signs of air and fluid such as were present in the first two patients reported in this series and who were in the hospital at this time. Dullness merging into flatness and diminution of breath and vocal sounds were present for about three weeks. Because of the child's continued improvement, no surgical measures were instituted and he was discharged on May 18. At this time, x-ray examination showed a disappearance of the fluid, a localization of the pneumothorax to a small area and the presence of a thickened pleura.

A follow-up x-ray film on Feb. 2, 1931, showed that both lung fields were clear with no evidence of any pathologic condition in the chest.

COMMENT

During a course of pneumonia, spontaneous pneumothorax may be produced by any of the following conditions: softening of the lung tissue, rupture of a subpleural emphysematous bleb, rupture of empyema through the lung abscess, multiple abscesses due to various organisms, circumscribed pneumonic processes leading to abscess formation and perforation of pleura. The exciting factor may be any act which is capable of increasing intrathoracic pressure such as coughing, sneezing, vomiting, hiccoughing or increased muscular strain.

It may be difficult to estimate the time relationship of the development of pneumothorax to that of the original pneumonia. Johnson[1] considered that there was a range of from four to twenty-five days with an average of ten and one-half days from the onset of the pneumonia in eight of his patients who developed the condition while under his observation. Based upon the time of appearance of either the first clinical symptoms or the characteristic x-ray findings, we may state that in our series, the pneumothorax occurred approximately on the eleventh, fifteenth, fifteenth, and seventeenth days after the onset of the original pneumonia.

According to the textbooks, the onset of pneumothorax due to any cause is generally sudden and acute in half the cases, and is accompanied by a sharp pain in the chest which is followed by a sense of suffocation, air hunger, cyanosis, severe coughing and orthopnea. This

dramatic picture is the exception instead of the rule in pneumothorax occurring during the course of pneumonia in children. It was observed in only one of our patients and in none of Johnson's[1] or Stoloff's.[2] In conformity with these authors we noted that the condition came on insidiously with a minimum of signs and symptoms, being recognized only by the alteration in physical signs from day to day or upon routine fluoroscopic or x-ray examination. Stoloff[3] states that "it (the insidious onset) is most apt to be so in the absence of complications. It is surprising how frequently simple pneumothorax, if not complete, may elude clinical recognition because of failure to produce physical signs commensurate with its extent." Our experience would tend to add pyopneumothorax under certain conditions to the above statement.

The signs and symptoms depend upon the degree of pneumothorax, the presence or absence of fluid, the amount of fluid, the condition of the pleura, the existing pathologic condition in the lung, and the presence or absence of mediastinal displacement.

Small, localized areas of pneumothorax may not result in any marked alteration in the shape of the chest wall, but greater involvement produces a perceptible fullness or bulging over the affected side. There are a lessened respiratory excursion and a lagging of the chest on that side. Cyanosis and dyspnea may be present but may be misinterpreted as being due to the empyema or pneumonia. Orthopnea is described as a prominent symptom, but it was noted in only one of our patients.

Percussion will give a hyperresonant, tympanitic, dull or flat note depending upon the proportionate amounts of air and fluid. The presence of considerable fluid, thick pus or marked thickening of the pleura with adhesions may mask and overshadow the signs of the air. This was exemplified in two of our patients. The first intimation of pneumothorax is either an increase in the areas of dullness or flatness or a change to hyperresonance or tympany. An increase in the areas of dullness and flatness was noted in all of our patients and hyperresonance in three.

Auscultatory findings depend upon the size of the tear in the pleura, the freedom of communication with the bronchus and the condition of the lung itself. The first intimation is a diminution and then an absence of breath sounds over a large area suggesting either pus or air. The voice sounds are also diminished or lost; the breath sounds are never vesicular. The typical characteristic findings of amphoric breathing, positive coin test, metallic tinkle and succussion splash clinch the diagnosis of pyopneumothorax but they are not always present. The succussion splash is not obtained in the absence of fluid or in the presence of thick pus or extensive adhesive pleurisy. We were able to elicit all of these signs in only one of the patients in this

series and in another child who developed pyopneumothorax subsequent to aspiration. Amphoric breathing was found in only two of our patients.

Displacement of the heart and mediastinum was noted in three of our patients. Displacement of the mediastinum is due to a disturbance of the normal physiologic intrathoracic equilibrium. The degree of the displacement depends upon the flexibility of the mediastinum, the amount of intrathoracic pressure in the pneumothorax area and the relative fixation of the mediastinal contents. Displacement does not occur in most cases because the mediastinum is fixed or practically fixed by adhesions or pleural thickening. Small areas of localized pneumothorax do not as a rule produce any displacement.

Two of the children complained of pain on the affected side for days after the onset. Pain was present even between attacks in the boy who had two recurrences.

The cough is as a rule nonproductive except upon rupture of an empyema or a localized pus pocket into a bronchus.

Roentgenographic diagnosis may be made by both fluoroscope and film. It is important that these be taken in both the erect and prone positions. The fluoroscopic examination in the erect position, if the child's condition permits, is the method of choice as the patient may be moved about and the fluid levels shifted.

The appearance varies with the amount of fluid, air, degree of collapse of the lung and the presence or absence of adhesions. Air shows as a clear sharply defined area of increased radiability in which there is complete absence of lung tissue. Fluid is shown as a dense shadow at the lower part of the chest with a horizontal line uppermost separating it from the air above. The fluid level is altered with a change in the position of the patient. The mediastinum, heart, esophagus and trachea may be displaced away from the affected side. The lung itself is collapsed and is visualized flattened against the vertebral column. There may be a narrowing of the intercostal spaces on the affected side.

Unilocular involvement shows an area of varying size which is sharply circumscribed from adjacent lung tissue by linear shadow. Multilocular pneumothorax gives a weblike or fish-net appearance.

The simple form probably does not have an adverse effect either on the course or on the outcome; on the contrary, the recent reports of Duken[6] and others,[7, 8] tend to show that the production of artificial pneumothorax in protracted pneumonia has a favorable therapeutic value. A similar opinion is expressed by McEnery and Brennemann[9] in their statement that pyopneumothorax is a help rather than a hindrance to a speedy recovery. The consensus of opinion, however, in the literature seems to be that pyopneumothorax in children has a deleterious influence on both morbidity and mortality.

The length of stay in the hospital in three of our cases (twenty-two, twenty-eight and twenty-nine days) was longer than that of uncomplicated pneumonia but shorter than that of empyema without pneumothorax. Packard[10] states that in his series of 50 children with empyema, the average duration of drainage was 46.1 days and that the average duration of the disease previous to operation was three weeks. X-ray examination at the time of discharge in all of our patients showed a partial pneumothorax without signs of fluid. Absorption of the air in two of these was noted in follow-up x-ray examinations taken one year later; another had two recurrences within two years and the fourth child could not be traced.

We found a mortality rate of 59 per cent in 41 cases in the literature of pneumothorax complicating pneumonia in children including those reported here. This is the same ratio found by Stoloff[2] in his study of childhood pneumothorax due to all causes. Not included in these statistics are 12 cases of spontaneous pyopneumothorax out of 94 consecutive cases of empyema in children mentioned by McEnery and Brennemann.[9] None of these 12 died, but 8 deaths occurred in 42 patients with pyopneumothorax following aspiration. All of our patients recovered, but another child, not included because the pyopneumothorax occurred after aspiration, succumbed.

TREATMENT

We do not know of any method of treatment which will prevent occurrence of pneumothorax during the course of pneumonia. Severe coughing and other acts producing an increase in intrathoracic pressure should be controlled. When pneumothorax has already set in, treatment may be considered from three angles: the immediate, during the course, and subsequent.

Immediate: opiates should be administered for the relief of pain and cough and measures instituted against shock.

During the course: As it is necessary for the fistula to close before the lung can expand to the normal, the patient must be kept quiet and cough controlled. Deep inspiration tends toward the aspiration of infected material into the pleural cavity. No other active treatment is indicated in simple noncomplicated cases or those with small amount of fluid but some advise strapping of the affected side. In moderate pyopneumothorax adequate drainage is essential; the pus may be withdrawn by repeated aspirations to be followed if necessary by closed thoracotomy in infants and younger children, or rib resection in older. We prefer the closed method. The removal of fluid and the subsequent injection of nitrogen have been advised by some.

Subsequent: The air may be withdrawn by repeated aspirations. In the valvular type, the valvelike formation permits the air to enter

the pleural cavity on inspiration but prevents its escape by closing on expiration. Cohen[11] has recently advocated and employed repeated suction by means of a special apparatus. Expansion of the lung may be accelerated by blowing into empty bottles. The child's general condition should receive constant attention. Orthopedic treatment is indicated in scoliosis and retraction of the chest.

SUMMARY AND CONCLUSIONS

Spontaneous pyopneumothorax complicating pneumonia in children probably occurs with more frequency than one would infer from the meager literature on the subject. Four cases are reported in children ranging in age from one to seven years. They all recovered but one had two recurrences in two years.

The classical picture of an acute onset is the exception in this type. It was present in only one of our cases. Pyopneumothorax may elude clinical recognition because of its failure to produce physical signs commensurate with its extent. The most constant and suggestive signs in our cases were diminished or absent breath sounds with dullness and flatness over an extensive area.

The importance of routine fluoroscopic and x-ray examination in the course of a pneumonia in infants and children and the careful daily observation of slight changes in the physical signs are stressed.

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ILEOCECAL (ENTEROGENOUS) CYST

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WHILE reviewing the literature on the subject of enterogenous cyst, we found it to be a comparatively rare phenomenon, only 28 cases having been reported.

History.—R. A. L., a boy, has been under our care since the age of two months. During this period the family and patient's history were essentially negative. At six months of age, he was brought to the office for examination on account of loss of appetite and irregular vomiting of feedings for the last three days. He had had an occasional stool with tenesmus, and a great deal of mucus but no blood.

Physical Examination.—The child was a well-developed, well-nourished male infant, apparently hungry, weighing 17 pounds 2 ounces and had a temperature of 101° F. The physical findings were negative except that the abdomen was distended with no rigidity of the muscles. There was a palpable mass, freely movable, about the size of an English walnut, globular in shape with smooth surface and quite a firm feel in the right lower quadrant. The child was sent to the hospital for observation with diagnosis of partial obstruction.

Radiologic Examination of the abdomen revealed evidence of increased density at the site of the palpable mass.

The erythrocyte count was 3,850,000, with hemoglobin 70 per cent; leucocytes 8,900 with differential of polymorphonuclears 47 per cent, small lymphocytes 50 per cent and monocytes 3 per cent.

Clinical Course.—At operation the terminal ileum was found to be greatly distended and the colon collapsed, with obstruction at the ileocecal valve caused by a tense, thick-walled cyst about 5 to 6 cm. in diameter. On attempting to enucleate the cyst its wall was broken with the escape of a fairly thin, clear, but definitely mucoid material. After emptying the sac it was possible gently to force the contents of the obstructed ileum through the valve into the cecum. The cyst was apparently situated between the walls of the terminal ileum lying immediately external to the mucous membrane and just above the junction of the ileum with the cecum. The mucous membrane bulged into the cavity after emptying the sac. The greatly distended ileum had spread out over the cyst wall to such an extent that it was impossible to resect the cyst wall completely without damaging the bowel. As much of the cyst wall as was possible was resected. Apparently no damage was done to the bowel and a fairly good lumen was found at the ileocecal valve. The postoperative progress was very satisfactory and the child was discharged as cured twelve days after operation.

Eighteen days after leaving the hospital the child was returned with the same symptoms of vomiting, loss of weight and every evidence of obstruction. A hard, round, smooth mass about the size of a golf ball, freely movable, was palpable in the right lower quadrant of the abdomen. At operation the terminal ileum was found somewhat dilated as a result of the firm mass within the lumen at the ileocecal valve. The appendix was long, thickened and appeared to be subacutely inflamed. The lower 3 inches of the ileum, the appendix, cecum and about 2 inches of the ascending colon were resected en masse, and a lateral anastomosis was

done. The pathologic report on tissues removed at the first operation was mucocele of the intestine. Examination of tissues removed at the second operation showed chiefly an excess of ileocecal tissue with more than the usual amount of lymphoid element.

The postoperative condition of the patient was excellent and an uneventful recovery was made. The child was discharged as cured in thirty days. We have examined him at monthly intervals since then and no deleterious effects of this experience have been observed.

MEDICAL ARTS BUILDING.

MONGOLOID IDIOCY IN A NEGRO INFANT

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IN THE white races mongoloid idiocy comprises about 15 per cent of all the mentally defective. Langdon Downs, in 1866, first attracted attention to mongolism, and from that time until 1922, when Trumpeer reported a case occurring in a Chinese boy, it was thought to be peculiar to the Caucasian race. Brousseau has listed twenty-eight countries and races in which it has been found. To these must be added a case in a Mexican; and, as it has been reported in the Scottish and Irish people, which she did not include, the total should really be thirty-one races.

Crookshank, in his latest book, denies the possibility of mongolism occurring in the negro, but Bleyer,[1] in 1925, reported three cases of mongoloid idiocy in negroes.

In a more recent article Bleyer[2] reports that in a study of fifty thousand dispensary patients in St. Louis, mongoloid idiocy occurred in about 14 per cent of all the mentally defective. He states that these figures are more nearly correct than those from institutions for defectives. These later figures vary so much as to be of little value. In this study he states that they have encountered eight cases of mongoloid idiocy in the negro race. He does not give the percentage of colored patients attending the clinic, but thinks the proportion of mongolism in the colored race is about the same as it is in the mentally defective of any race.

In the out-patient department of Baylor University in Dallas, Texas, we have a colored attendance which is at least double that of the white people. In the last five years, no case of mongoloid idiocy in negroes has been recorded, while an average of three to four a year has been encountered in the white race. In talking to doctors, who have been in practice in this locality, heavily populated with negroes, none have admitted ever seeing a case of mongolism in a negro.

I wish to report a case of mongoloid idiocy, occurring in a seven-month-old negro male infant.

CASE REPORT

The infant was admitted to the hospital because of an acute upper respiratory infection. At this time it was first noted that he had all the classical signs of mongoloid idiocy; the bullet-shaped head, the slanting eyes, the small short hands and fingers with extremely short "little fingers" and very relaxed musculature (the legs can easily be extended back on the shoulders). He had a "blue spot" on one arm, which has been described in this condition. He never cries and cannot sit alone at seven months. The mother, twenty-five years of age, has the physical appearance of a full-blooded negro, but she states that her father's mother was said to have some Indian blood. The father is twenty-six years of age and is not a full-blooded negro, but he does not know how far back the mixture of blood occurred nor whether it is with white or Mexican blood. The marriage has resulted in one other previous pregnancy—a five-year-old normal girl.

Fig. 1.—Photograph of seven-month-old negro infant with mongoloid idiocy.

SUMMARY

A case of mongoloid idiocy in a negro infant is a great rarity in a locality where the majority of patients seen in an out-patient department are colored children, either full blooded or a mixture of negro and other races. The family history in this case would suggest some such mixture. It is exceedingly rare and the incidence of mongoloid idiocy in either pure or mixed negroes does not seem to approximate that seen in mental defectives in the white race.

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ANTIQUES OF PEDIATRIC INTEREST

T. G. H. Drake, M.B., F.R.C.P.(C.)

AMULETS have been defined by Petrie as "Objects worn by the living without any physical use but for magical benefits, or placed with the dead, or set up in the house for its magical protection, apart from deities for household worship." These amulets are of small size and usually bear a loop for suspension about the person—the neck, chest, limbs or waist. They were worn by the living and buried with the dead.

Egyptian Amulets

1.—Papyrus sceptre, for protection during youth, 500 B. C., green glaze, length 8 cm.
2.—Bes, the god of children, dances and games, 500 B.C., blue glaze.
3.—Taurt, 500 B.C., goddess of pregnancy, green glaze.
4.—Isis and Horus, 500 B.C., blue glaze.
5.—Shrewmouse box for protection against boils, 1000 B.C., metal.
6.—Scaraboid for quartan fever, 500 B.C., white glaze.
7.—Snake box for easing delivery, 1000 B.C., metal.

The material from which they were manufactured was glazed earthenware, especially green and blue, glass, bronze and various ordinary and semiprecious stones.

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Among the 270 different Egyptian amulets listed by Petrie the following are of pediatric interest. The meanings of these amulets as given by him have been taken from various sources—the *Book of the Dead*, and references from other countries, especially Pliny, where no reference to their meaning is obtainable from ancient Egyptian sources.

Breast, power of lactation.

The frog or toad, symbolizing Heqt, the goddess of birth.

The papyrus sceptre, signifying Youth.

The wolf's tooth, for dentition.

The dolphin's tooth, against infant's fright.

The scarab with legs, for protection against quartan fever, the horns of the scarab for children.

A cobra resting on a box-like case, for easing delivery.

The Amphisbaena, or Taurt, on case, for protection during pregnancy and easy delivery.

Coral worn by infants.

A bell worn on a bracelet as protection against the evil eye.

Isis and Horus, for the protection of Isis.

Bes, the god of children, of dances and of games.

Shrewmouse, for protection against boils.

The hippopotamus, sacred as Taurt, the goddess of pregnancy.

The material of which the amulet was composed was also significant. Crystal assisted in parturition and malachite safeguarded the infant. White stones increased the supply of breast milk. In modern Italy, white chalcedony, alabaster and white coral are still used by the nursing mother to ensure successful nursing. In addition, frequently the modern Italian child is protected against the evil eye by amulets of shells, clams, horn, teeth, toads, crescents, crystal, agate and sexual models. The idea is that the dangerous thoughts of another person may be diverted by the wearing of some object which distracts the attention.

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Critical Review

THE NEWBORN

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THOSE interested in the newborn have realized for a long time that in this particular field of medicine lies the greatest opportunity for reduction of the mortality rate. To accomplish this it will be necessary to stimulate intensive research in problems of the newborn and to increase the number of hours devoted to study of neonatal conditions in medical schools. Many of the incorrect diagnoses of the cause of neonatal death made by physicians are due to self-admitted ignorance from lack of adequate instruction. Williams[1] states that if sums as large as those spent in trying to find the cause and cure of cancer were to be spent in investigating problems of the newborn the resultant saving of life would be infinitely greater and yield even more satisfaction than it would in the older age group. Regarding instruction in medical schools, the report of the subcommittee on Pediatric Education of the White House Conference on Child Health and Protection[2] shows that half of the schools investigated devote less than ten hours to study of the newborn. Erlich[3] finds a similar situation in France and urges that the curriculum of medical schools be arranged to give more time to neonatal conditions.

NUTRITION

The subject of neonatal nutrition must include consideration of fetal nutrition as well. The fetus utilizes the same organic and inorganic substances as the older infant or adult but its supply is dependent upon the placental barrier which normally is open to the necessary requirements and closed to the unnecessary or harmful ones. The fetus is considered a parasite. However, its nutritive supply seems to be governed in some way, possibly by a hormone,[4] for fluctuations of fats, proteins, and carbohydrates in the maternal diet do not seem to directly affect the size and weight provided there is not an actual deficiency. There has evolved a somewhat different attitude in respect to the effect of the maternal mineral and vitamin intake upon the fetus. While the average American antenatal diet is rarely deficient in fat, protein, and carbohydrate, there is probably a mineral and possibly a vitamin deficiency. This has led to the assumption that the fetus may suffer because of lack of these substances. Such a conception, as far as calcium is concerned, has not been substantiated by estimations of the calcium concentration of the blood serum of newborn infants, nor by the studies of Booher and Hansmann[5] who found a practically constant degree of calcification of the skeleton of the newborn regardless of the maternal intake of calcium, although some of the mothers showed evidences of calcium drain. While these investigations serve to corrob-

orate the parasitic characteristics of the fetus they do not necessarily refute the idea that deficiencies of the maternal diet do not have a good deal to do with the subsequent development of rickets, abnormal dentition, or other manifestations of calcium deficiency. In lieu of the tendency of the human organism to utilize minimal amounts of mineral necessities when there is a scarcity of them in the diet, although physical signs of this may not be evident,[6] it is possible that a deficient maternal diet may produce a poor fetal mineral reserve with later evidences of insufficiency. Thus the storage of phosphorus and calcium may suffer with development of postnatal rickets. It is, therefore, probable that the reserve of minerals and possibly of some vitamins can be augmented by an adequate maternal diet. It also seems possible to influence metabolism by diet so as to prevent maternal injury from mineral and vitamin deficiency, and to produce an offspring better able to cope with the conditions of postnatal life.

Birth weight has been found to be dependent upon many factors, chief of which are race, fetal maturity, season of the year, and the condition of the mother during pregnancy. The latter includes maternal health, the number and the interval between pregnancies and gain in weight during pregnancy.[7] The relation between maternal gain and birth weight is still a matter of controversy. Gerschenson[8] found that during hunger years the percentage of babies with low birth weights was nearly twice as great as in years of prosperity. These findings are not in accord with some American views where the question is raised whether or not diet plays much part in birth weight.[4] Others are unable to decide this point.[9] This may be due to the fact that up to now there has been little opportunity in this country to make observations under conditions of food shortage. Reports of the relation between birth weight and minimum balanced diets will be watched for with interest, and I cannot help but feel that such a relationship will be found. Regardless of the controversy over the effect of excessive or minimal diets upon birth weight, present conditions are such that an adequate well-balanced diet ration should be assured every pregnant woman.

The average initial loss of weight after birth is about eight ounces. Attempts have been made to prevent or lessen the initial weight loss as well as to hasten the return to birth weight by early institution of feedings of some sort. Breast milk or cow's milk supplements, lactose water, and even alkalis, such as sodium citrate have all been tried with varying success. Placing the infant to the breast of mothers who have very young babies has not been successful. In fact, it has often aggravated the loss. Oeljen and Scherer[10] found that those infants who were not put to breast for twenty-four hours showed a smaller loss of weight than those put to breast earlier. Furthermore a greater percentage of infants put to breast late regained or exceeded their birth weight by nine days. Bottle feeding of breast milk or formula has been of some value but as a whole infants do not take food well the first day. After this period complementary feedings give better results. Dwyer and Neff[11] have found that under careful supervision such feedings result in a more rapid return to birth weight. They believe that it is better to have the baby leave the hospital obtaining sufficient food to be contented and thriving if it can be done without endangering the breast supply. This means that a complementary should not be given

provided the breast supply is adequate, and if this is not the case, it should be withheld until the infant has emptied the breast. Such a procedure will not decrease the breast supply nor will it hasten weaning. However, careless methods such as are carried out in many private hospitals lead to early weaning. While the advance in feeding methods and the development of simple formulas has brought a greater safety element into artificial feeding, the general population gets little experienced advice. Artificial feeding, therefore, is not as safe as would be suggested by their findings. Infants of normal weight who made satisfactory gains received about sixteen ounces of milk each day which was estimated as 47 to 50 calories per pound of body weight. From personal experience as well as the report given above, it is apparent that breast feeding may be jeopardized by careless use of complements or supplements. While the statement of Brennemann[12] that there need be no fear that the baby may show preference for the bottle is true as far as taste is concerned, the danger lies elsewhere. Retrogression of the breast supply with the necessity for greater effort upon the part of the baby to obtain the milk will frequently cause it to give up trying. Elder and Bakewell[13] have reported some success (average loss 7 ounces) by giving two ounces of 5 per cent lactose water every four hours. When sodium citrate, grains V to every two ounces, was added to the lactose solution the loss was reduced to five ounces. The infants so treated regained their birth weight earlier, few had inanition fever temperature, and their general condition was better.

Such procedures are not new. They can be compared with the old-time habit of giving honey water or albumin water. The whole problem resolves itself about the question, "When should the birth weight be regained?" There seems to be a generally conceded opinion that the arbitrary limit is ten days. There is no lucid reason why all babies should regain their birth weight on this day, for there are too many individual factors to be considered. A few of these are: type of delivery, trauma of birth, reaction to the first few days of life, time of onset of lactation and the sufficiency of breast milk, the care the baby receives, and its maturity. The misguided idea of arbitrarily fixing the time when birth weight should be regained is comparable with other questionable attitudes about infant weight upon the part of mothers and has had the unfortunate result of making them too weight-conscious. Even more descriptive is the term, weight-anxious. The usual reaction to this is overfeeding with its harmful effects upon the baby, especially if the infant is artificially fed or weaning. The answer to the question cannot be made more definite than to say that probably by the end of the second week the infant should be at or about its birth weight.

In spite of the arguments in favor of adoption of measures aimed at preventing weight loss or bringing about a quick return to birth weight, the reviewer is not convinced of the urgency of such attempts. In the first place, the loss which is to be regarded as physiologic, is due to water loss from insufficient intake. There also occurs a small tissue loss, a decrease in the glycogen reserve, and an outflow of some minerals. Many of the chemical constituents of the blood become concentrated. Thus there is an increase of blood chlorides.[14] The explanation for the mild compensated acidosis found during this period of inanition and

dehydration is thought to be due to the excess of chlorides rather than the loss of available base.[15] Under normal conditions tissue loss is so negligible and acidosis so mild and evanescent that neither requires much attention. In the second place, while all of the ferments of the gastrointestinal tract are present in amounts sufficient to permit digestion to take place immediately after birth, this tract whose activity has been held in abeyance in utero is easily upset in the early days of life. This is manifest in the frequency of the so-called but little mentioned initial diarrhea of the newborn and other evidences of intestinal irritation. The gradual administration of food is more natural and less likely to cause irritation. In the third place it is a well-established fact that the newborn period is one in which the intestines of many infants have the power to absorb undigested protein, and thus possibly develop protein sensitization. The use of albumin water or modified milk complements is probably a factor in the development of allergy. Such conditions as eczema, colic, urticaria, and asthma have been attributed to sensitization in the early days of life to some protein. The fact that the casein of cow's or goat's milk may also sensitize the infant to milk of its own species[16] makes it obvious that the generous use of such supplements is not without its dangers. Possibly some of the so-called cases of congenital allergy may originate in this way. And in the fourth place the effect of early suckling may possibly have a deleterious effect upon minor birth injuries which would be benefited by an early rest period. The difficulty of getting an infant to nurse properly is on the face of it suggestive of intracranial injury.

It can be seen, therefore, that the greatest necessity at this period is water. It is very likely that the amount of water taken is largely responsible for the results attributed to various solutions. Furthermore, during the course of such studies more careful attention is given the infant so that the intake of fluids is greater than normal. In respect to the use of honey water, lactose water, or albumin water the caloric value of the substances given is probably not more than 60 calories, which is barely one-third of the basal metabolic needs (62 calories per kilogram per 24 hours). As far as the use of sodium citrate is concerned the only possible influence it might have upon weight gain would be to favor retention of fluids although it possibly might be a factor in decreasing the mild acidosis. The necessity for this is not apparent since the acidosis is not due to a deficiency of base.

When lactation is delayed or through some other fault there is a continued loss of weight, complementary feedings are indicated. Usually a loss over eight ounces demands such feedings. Human milk is preferable when the supply is known to be pure but often it is unobtainable. Cow's milk is the next choice. It should be heat treated for several reasons. Boiling, the evaporation, or drying processes are known to make the curd more digestible while at the same time they do away with the danger of bacterial contamination. It has been found also that the tolerance of allergic infants is greater for heat-treated milks than for raw or pasteurized milks.[17] Recently in some localities there has been an attempt to extol the virtues of soft curd milk, that is milk with a low curd tension. Aside from the fact that boiling or other methods of heat treating make milk safer bacteriologically they also

affect the curd in such a way as to make it more digestible. It has been found that the percentage of casein seems to be the great factor in influencing curd tension. Milk of high curd tension means a more concentrated milk and therefore one of more food value.[18] Infants fed soft curd milk need a higher intake than those fed other milks. Elias,[19] studies confirmed the remark of Brennemann in 1913 that test tube experiments do not actually reveal what goes on in the infant's stomach. From the numerous reports at hand there seems to be no objection to the use of evaporated or dried milks,[20, 21] in fact from the standpoint of the allergic child it would seem to be indicated. Processing does not destroy vitamins A and B.[22] Acid milks seem to be well tolerated by the newborn[23] and have been recommended by many. According to Gerstley[24] whole lactic acid milk is apparently as easy to digest as breast milk although the reaction of the intestines to this type of feeding does not differ from plain cow's milk feedings. It is also a food likely to lead to overfeeding. He raises a question as to the value of acidulated milks. The usual cow's milk formula for newborn babies consists of boiled milk one-third, boiled water two-thirds and about one dram of dextri-maltose to three ounces of mixture. Evaporated or dried milks may be used in place of fresh milk. For many years we have used equal parts of milk and water with no apparent difficulty. Sauer[25] recommends even a slightly stronger stock formula consisting of evaporated milk, six ounces, water twelve ounces, and lactose one ounce. This has a caloric value of about twenty-one to the ounce. These mixtures with a higher nutritive content have shown no signs of irritating the alimentary tract and have produced more satisfactory gains.

Emanuel's[26] observations on nicotine in the breast milk of cigarette smokers may be of interest to some. Nicotine could be demonstrated after seven cigarettes were smoked but no ill effect was noticed up to fifteen. As one would expect lactation apparently was uninfluenced by smoking.

Bearing in mind the fact that many babies cannot be given constant pediatric attention, the whole problem of satisfactorily caring for its nutritional needs in the early days of life may be summarized as follows:

1. Put to breast once in the first 24 hours.
2. In order to avoid unnecessary damage to nipples restrict the nursing time to 3 to 5 minutes until lactation is well established.
3. Fluids equal to one-sixth of the body weight should be given daily. (16 to 20 ounces.)
4. Five per cent lactose water may be used during the inanition period.
5. A four-hourly interval is usually sufficient. Omission of one night feeding is often possible.
6. Complementary feedings should be instituted when the weight loss is over eight ounces.
7. Breasts must be emptied before complementing to prevent loss of milk.
8. About 16 ounces of heat-treated milk formula (boiled, evaporated, or dried) will satisfy the caloric demands of 45 to 50 calories per pound per 24 hours. (See formulas given above.) Avoid overfeeding.

9. Vitamins C and D must be supplied relatively soon in the form of orange or tomato juice and cod liver oil or viosterol.
10. The assumption that birth weight is usually regained in two weeks will make mothers less weight-anxious.

ALIMENTARY TRACT

The recent literature relating to neonatal disorders contains the usual profusion of case reports of congenital anomalies. To the casual reader these might seem to be of purely academic interest but upon careful analysis it is evident that a clearer understanding of the whole subject might bring about the saving of a few lives that would otherwise be lost. Vomiting is one of the most common symptoms of the newborn infant. Since early recognition of its cause is essential if corrective measures are to be instituted while there is a fair chance for success, it seems timely to review the many articles referring to the alimentary tract from the standpoint of this prominent symptom.

The most frequent form of vomiting at this age is regurgitation. It occurs at irregular intervals and is probably due in most instances to belching of swallowed air, irritation of ingested food, or overfilling of the stomach. The condition usually is a transient one which responds readily to simple measures such as ejection of the air bubble immediately after nursing by patting the infant's back, reduction in the size of the feeding or lengthening the feeding interval. When, however, vomiting is a more constant aftermath of nursing, is projectile, or associated with progressive dehydration and inanition an immediate attempt should be made to ascertain the cause. No credence should be given the old idea that persistent vomiting during the first weeks of life is due to lack of tolerance to food. The oft-heard complaint that nothing can be found to agree with the baby characterizes an attitude upon the part of parents that has led to the death of many infants whose lives might have been saved by prompt diagnosis and treatment. The most common instances of vomiting urgently demanding attention are those caused by obstruction of some sort. The obstruction may be functional or organic.

VOMITING FROM FUNCTIONAL CAUSES

The functional disorders consist of single or multiple spasms located in various parts of the alimentary tract. They sometimes produce symptoms of sufficient severity to suggest obstruction of an organic nature. In general they seem to be associated with a congenital deficiency of the autonomic nervous system, characterized chiefly by irritability of the vagus nerve. Spasms of this type are included in the group of functional disorders called vagotonia by Eppinger and Hess.

Esophageal spasm is rarely found unassociated with spasm of the cardia or pylorus. It is probably a cause of vomiting early in life but usually the symptoms do not become pronounced until a later age. Undoubtedly fluoroscopic examinations of newborn infants with vomiting of an obscure nature would yield more frequent diagnoses of this condition.[27]

Cardiospasm seems to have escaped the notice of most clinicians until Birnberg[28] and Segar and Stoeffler[29] recently reported five cases. The existence of this condition from birth is apparent from the history of

cases of cardiospasm in older infants. It is characterized by forceful vomiting appearing with the first and subsequent feedings, rapid loss of weight, and dehydration fever. No gastric contents are found in the vomitus. The diagnosis is confirmed by roentgenologic examination which reveals delayed passage of the opaque substance through the cardia in case of mild spasm and complete or nearly complete obstruction in the severe cases. Treatment consists of dilatation and feeding by catheter, which serves to relax the spasm in most cases and keep up nutrition. No doubt the ease with which catheter feeding has overcome repeated vomiting during the first few days of life has led most of us to fail to recognize the true cause. In some cases, especially those associated with congenital shortness of the esophagus and roentgenologic evidence of a high thoracic stomach, dilatation by bougie is necessary.[30]

Pylorospasm is the most frequent form of functional disturbance of the digestive tract. It is considered by many to be associated with spasticity of the intestinal musculature, the so-called syndrome of gastroenterospasm. Others believe pylorospasm merely a stage in the development of congenital hypertrophic stenosis of the pylorus. There is no need to go into the details of this controversy for the most important thing is to recognize the fact that the affected infant is suffering from an obstructive disturbance requiring prompt attention. The symptoms are those of projectile vomiting appearing shortly after feeding, constipation, oliguria, progressive inanition, and later occurrence of acidosis or alkalosis.[31] The vomitus contains no bile. Peristaltic waves of moderate intensity are seen passing from left to right over the upper abdomen. These begin after feeding and continue until the stomach is emptied. Of diagnostic value is the fact that these symptoms appear in the first few days of life while those of hypertrophic stenosis are more likely to develop in the second or third week. Infants with such symptoms should be placed upon a well-organized course of medical treatment to determine whether or not surgical intervention is necessary (see pyloric stenosis). Atropine sulphate 1–1000 solution, is administered orally five times a day, preferably one-half hour before feeding. It must be remembered that the drug should be given in sufficient doses to obtain the proper effect—physiologic flushing of the skin. The dose varies from one to three and occasionally five drops, for infants' tolerance to atropine varies greatly. No doubt some of the failures have been due to insufficient dosage or possibly to the fact that spasm was only a minor consideration as compared with hypertrophy of the pylorus. The feeding interval should be lengthened to four hours. Gastric lavage is often of value in removing mucus. Breast milk is the feeding of choice but where this is not possible, a concentrated or even thick cereal feeding may be used with success. The gruel should be at least thick enough to necessitate enlarging the nipple openings to one-quarter of an inch.[32] Gavage feedings are necessary sometimes. If a trial period of a week results in definite but gradual diminution of vomiting, gradual gain in weight and passage of large stools, recovery usually is complete in six weeks to three months. Infants not responding to this treatment should be referred to the surgeon. This is especially true if a progressively growing tumor mass is felt in the right hypochondrium. The longer such infants are permitted to go untreated the graver the prognosis. Other symptoms of gastroenterospasm are restlessness and discomfort after feeding, hypertonicity

of the skeletal muscles, and constipation or diarrhea. This baby is said to be nervous or have colic. Prompt response to suitable doses of atropine sulphate suggests that this condition is due to an imbalance of the autonomic nervous system.

It is difficult to classify one type of vomiting about which there has been considerable controversy. This is the so-called Tetany of the Newborn. Bass and Karelitz,[33] McGavran,[34] Nesbit,[35] and others have described a syndrome occurring in the first few days of life which is characterized by projectile vomiting and dysphagia, together with the usual signs of tetany such as carpopedal spasm, laryngospasm, convulsions and a positive Chvostek facialis phenomenon. Mitchel[36] points out that too much credence should not be placed upon the presence of a Chvostek sign at this age since he found it consistently present in 55 apparently normal infants. Diagnosis of tetany, therefore, should not be based solely upon the presence of this sign, but should include determination of the reaction time to the galvanic current, a thing that was not done in some of these cases. However, there are indications that tetany is present at times in the newborn, but in such instances it is probably not associated with rickets,[37] except perhaps in some prematures.[38] The controversy over tetany in the newborn seems to have come from belief that there must be an associated rickets or a low serum calcium, and that the tetany is primary. Obstructive vomiting may produce alkalosis by reduction of plasma chlorides and a corresponding increase of plasma bicarbonate.[31, 39] As a result of this there may be a reduction of serum calcium with manifestation of tetany. However, a low calcium concentration is not absolutely necessary if the pharmacologic activity of calcium is assumed to be due to the ionized fraction, for it is possible to have a reduced calcium ion concentration with a normal serum calcium content. Tetany in the newborn is more likely to be the result of vomiting than the cause of it. Kramer who discussed the paper of Bass suggests that intracranial damage during labor initiates vomiting which in turn is responsible for tetany. Certainly such phenomena as dysphagia, hyperpyrexia, vomiting, and convulsions could be the result of intracranial pressure. Whatever the cause, tetany-like symptoms are a therapeutic indication for calcium and viosterol administration.

VOMITING FROM ORGANIC CAUSES

Organic obstruction of the alimentary tract manifests itself as atresia or stenosis from faulty canalization, malposition, failure of rotation, and torsion, or from pressure exerted by aberrant vessels, abnormal bands, or tumor masses. When the obstruction is recognized as organic all that is necessary to know is its approximate site before attempting surgical correction.

Atresia of the esophagus occurs more frequently than is ordinarily supposed. In the last year I have seen two cases and in the past fifteen years a total of ten. Rosenthal[40] in 1931 reported eight cases which brought the number of reported cases up to 255. Thirteen more have been described since then.[41] In fully 75 per cent of the cases there was a communication between the esophagus and the trachea, usually the lower rudiment. The symptoms are those of persistent drooling with cessation of respiration, cyanosis, and choking when fluid is taken. This is followed by regurgitation of a frothy discharge through the nose

which subsides when feeding is stopped. The vomitus contains neither curds nor gastric juice. When a catheter is passed it meets with an obstruction at about 10 cm. from the alveolar ridge. Meconium may be expelled but milk stools are never seen. The infant becomes markedly dehydrated and starvation acidosis develops. The diagnosis can usually be made without the use of the roentgen ray although such examinations are very useful in determining the completeness of the obstruction or the presence of a fistula. When the stomach is found distended with air, a communication between the esophagus and the trachea should be immediately suspected. This can be verified by the injection of iodized poppy seed oil into the trachea. The presence of a fistula makes the prognosis hopeless.

Feeding through a gastrostomy tube only hastens death by aspiration pneumonia. Such feeding may be followed by ejection of curdled milk from the trachea causing the erroneous assumption that the esophagus is not completely closed. Even if there is no fistula aspiration pneumonia is likely. Thus it seems hardly justifiable to subject these infants to an operation, at least until better surgical procedures have been devised, unless there is good reason to believe that the obstruction is caused by a thin membrane. It has been suggested that intraperitoneal ligation of the cardiac end of the stomach and a gastrostomy might be successful, but even so pneumonia from aspiration of saliva would always be imminent.

Congenital hypertrophic stenosis of the pylorus is one of the types of obstruction most amenable to treatment. The recent papers of Lanman and Mahoney,[42] Donovan,[43] Page,[44] and McLeod[45] adequately summarize the situation from a surgical standpoint. The condition, which peculiarly enough is found largely in males, is characterized by five cardinal symptoms:

1. Failure to gain in the absence of fever or infection.
2. Persistent projectile vomiting of bile-free stomach contents shortly after feeding.
3. Visible gastric peristalsis, from left to right.
4. Scanty stools of the hunger type.
5. A palpable pyloric tumor.

Other confirmatory symptoms are found also. There is loss of fat, dehydration, ketosis or alkalosis with evidence of gastric tetany, fullness of the epigastrium with flatness of the hypogastrium, and sometimes diarrhea from the excessive use of cathartics and enemas. The symptoms usually do not date immediately from birth. Donovan found that the average was three weeks. Early vomiting is suggestive of spasm or duodenal atresia. The use of the x-ray is not essential and the presence of barium in the stomach is a handicap at operation. Lanman resorted to roentgenograms in only 6 of his last two hundred cases. Operative treatment is the method of choice of many physicians in whose hands it has given prompt and lasting relief of symptoms.

Safe surgical treatment rests upon close observation of many details:

1. Replacement of lost body fluids, by hypodermoclysis of saline, 3 per cent glucose, or by blood transfusion.
2. Maintenance of body heat during operation.
3. Rigid asepsis and thorough hemostasis.
4. Postoperative maintenance of fluid requirements.

Ether seems to be the anesthetic of choice of most surgeons at present, although we see no objection to the use of local anesthesia. The Fredet-Rammstedt operation of pyloromyotomy is the universally accepted one because of the simplicity and speed with which it can be performed. Blunt dissection of the fibers of the pyloric ring reduces hemorrhage which must always be controlled by suture. The mortality rate seems to be related to the experience of the operator. Thus Lenman and Mahoney lost 10.4 per cent of their first 125 cases, 7 per cent of the next 150, and 2 per cent of the last 150 cases. The mortality figures of others are very much the same. The reduction is due to development of greater operative skill and gentleness and to better methods of care before and after operation. Patterson[46] believes that a further reduction of the mortality rate may be brought about by isolating these highly susceptible infants from the infections of a general ward. These experiences emphasize all the more the necessity for correct diagnosis and for strict adherence to a well-regulated test-out medical treatment before subjecting these infants to the hands of less skilled operators. Some men still cling largely to the medical treatment reserving surgery for the more severe cases. Boone[32] states that the following should be operated upon without delay:

1. Children with severe vomiting and hunger stools.
2. Those out-of-town infants who cannot be closely observed while under medical treatment.
3. Children whose parents are not sufficiently intelligent or refuse to cooperate.
4. Infants less than six weeks old who are in a serious nutritional state.

He thinks that the following types may be safely given a trial period of medical treatment:

1. Breast-fed infants in fair condition, provided they respond by less vomiting and either stationary weight or a gain in three to five days.

2. Bottle-fed babies who although vomiting are still having small but definite stools.

If after a period of one or two weeks there is no definite gain in weight, vomiting is still frequent, and the stools remain scanty, operation should be resorted to. At present the conservative thought seems to consider treatment dependent upon the severity of the disease and the skill of the operator. Many of the milder forms may suffer at the hands of an inexperienced operator. Even Lanman and Mahoney admit, "... the apparent simplicity of the operation has been a source of considerable danger to these patients, for, as Barrington-Ward states in the preface to his *Abdominal Surgery of Children:* 'The adult may be safely treated as a child, but the converse can lead to disaster.'" The fact that certain children's hospitals treat practically all cases of pyloric stenosis surgically because of their enviably low mortality records does not justify such an attitude in localities where experience in the surgical treatment of this condition is considerably less. Under such circumstances medical treatment should be given a fair trial. Fully 50 per cent of the cases can be successfully treated in this manner. (See pylorospasm.)

Another cause of obstructive vomiting is occlusion of the intestinal canal. Over 500 cases have been reported excluding the most common form of all, stenosis of the rectum and anus. Stenoses of the ileum and duodenum are next in the order of frequency. The time at which the vomiting occurs after feeding determines to a large extent the site of the lesion. In duodenal atresia or stenosis it comes on early making differentiation from pyloric stenosis difficult. Other symptoms are abdominal distention, rapidly progressive dehydration and inanition, and absence or diminution in the number of stools. The evacuations contain no bile if the lesion is below the ampulla of Vater and jaundice may soon develop. If the lesion is high up abdominal distention is not as marked. Roentgen-ray examinations are of value but should be avoided whenever not essential for diagnosis since the presence of barium at operation is a decided handicap. It should be evacuated by gastric lavage before operation. A roentgenogram taken with the infant in the upright position is valuable in revealing levels of fluid with gas above. Such a finding with demonstration of loud borborygmus signifies obstruction. The prognosis is poor even if the obstruction is not complete and the fact that in 15 per cent there are multiple occlusions decreases the chances for successful surgery.[47] However, as Webb and Wagensteen[48] point out, there is some hope for these infants. Of the twelve successful operations, ten were for duodenal and two for lower intestinal obstruction. (Ladd,[49] Carter.[50]) Surprising as it may seem enterostomy does not seem to be the operation of choice, for none of the infants so treated have lived. The successful operations have all been anastomoses, even though such procedures are exceedingly difficult in such small patients. Preoperative and postoperative care is very important the same as for pyloric stenosis. In view of the fact that some successful operations have been recorded it is very possible that this number could be greatly increased if diagnoses were made more quickly.

More amenable to treatment is obstruction of the duodenum by transduodenal bands.[51] These either extend from the liver across the duodenum to the transverse colon or arise from the superior mesenteric artery.[52] Acute symptoms may appear in the neonatal period giving rise to a clinical picture simulating hypertrophic pyloric stenosis. The absence of a tumor mass helps to rule this out. When at operation pyloric stenosis is not found the sub-hepatic fossa should be explored for transduodenal adhesions or bands. If obstruction is localized in this region surgery is indicated immediately for the death rate is less when severe dehydration and inanition have not developed. Occasionally the obstruction is of a more chronic nature.[53, 54]

Atresia of the rectum or anus is the most common obstructive lesion of the intestinal tract. The diagnosis is usually simple. Nothing new in the way of surgical treatment has been recommended. Intestinal obstruction from other causes is extremely rare. Intussusception must be considered in the differential diagnosis but it is less common at this age than in later infancy. Falls and Jaffe[55] report four interesting cases of intestinal obstruction due to a collection of inspissated mucus in the rectum extending sometimes into the colon. Failure to pass meconium and symptoms of intestinal obstruction should lead to a careful examination. When the plug is short minor measures are sufficient to evacuate the rectum. When the obstruction is higher, and the

usual sites are the sigmoid or ileocecal regions, operation is indicated. It is not unlikely that some cases of this nature are left untreated because they are not recognized. Perforation of the intestine may follow this. While the causative factor of Hirschsprung's disease is often present at birth symptoms are not usually noticeable until later on.[56]

The table given below may be of value in differentiating between functional and organic types of vomiting as well as an aid to localizing the obstruction. The fact that roentgenologic examination is rarely necessary should make it possible for those who do not have x-ray facilities to make the diagnosis early.

DIFFERENTIAL TYPES OF VOMITING

	FUNCTIONAL	ORGANIC
Vomiting	Inconstant Often projectile	Constant Always projectile
Stools	Meconium and scanty milk stools	Meconium only
Alkalosis	Rare	Usual
Dehydration Inanition	Moderate or slight	Severe and rapidly progressive
Hypertonia	Early	No—Tetany late
Response to medical treatment (atropine, etc.)	Satisfactory	None

SITE OF OBSTRUCTION

	ABOVE STOMACH	ABOVE AMPULLA	BELOW AMPULLA	ABOVE ANUS
Vomiting time	During feeding	Shortly after feeding	After first feeding	None or very late
Vomitus	No curds No HCl No bile	Curds HCl No bile	Curds HCl Bile	Curds, HCl, Bile, Feces } if present
Stools	Meconium only Bile	Meconium Bile	Meconium No bile	Scanty meconium No bile—or none
Abdominal distention	None	None or epigastric	Moderate upper abdomen	Marked entire abdomen
Visible peristalsis and location	None	Epigastric	Upper abdomen	Entire abdomen

Above Stomach: Esophageal and Cardia obstruction.
Above Ampulla of Vater: Pyloric or high duodenal obstruction.
Below Ampulla: Obstruction low in duodenum, jejunum, ileum and cecum.
Above Anus: Obstruction in colon, rectum or anus.
Palpable tumor mass in hypogastrium in pyloric stenosis.
Sausage-shaped tumor mass at site of obstruction in intussusception.
Palpable tumor mass in right hypochondrium in pyloric stenosis.

OTHER ALIMENTARY DISORDERS

Some of the other disturbances of the digestive tract in which vomiting is not an important or common symptom consist of infections, malpositions, and hernias.

Abcesses of the salivary glands, which are unassociated with generalized sepsis are seen occasionally. Kriss[57] reports two cases in which the offending organism was the staphylococcus. The bacteria probably ascend from the mouth successfully setting up an infection in infants having a lowered resistance to this organism. I have recently seen a

similar involvement of one of the parotid glands. The possibility of obstruction to the lumen of the secretory ducts might favor infection from stasis. Treatment consists of warm packs followed by surgical drainage.

High thoracic stomach associated with cardiospasm and congenital shortness of the esophagus has been mentioned before. Other thoracic displacements of the stomach and other viscera are due to either an actual defect in the diaphragm (diaphragmatic hernia) or to a high diaphragm (miscalled eventration). The former are compatible with life if the defect does not permit massive herniation of abdominal organs into the thoracic cavity. In such cases the symptoms are chiefly respiratory. In the latter case the defect is usually found casually during fluoroscopic examination. Under such circumstances operative interference is not warranted. Most congenital diaphragmatic hernias are left-sided probably because the right side is protected by the liver. However, the recently reported cases of Patterson,[58] and Thursfield,[59] were right-sided. Usually there is no hernial sac and therefore the term hernia is a misnomer. The massive hernias are fatal.[60] It is interesting to note that x-ray evidence of massive herniation of the abdominal viscera into the thorax may be absent if the baby dies at or shortly after birth. Probably some of the unexplained instances of early neonatal death may thus be attributed wrongfully to obstetric judgment, if an autopsy is not performed (Newberger[61]). Congenital elevation of the diaphragm is more rare than hernia. It is due either to a failure of development of the diaphragmatic musculature or the phrenic nerve. Injury to this nerve during delivery of the after-coming head is said to be an etiologic factor. In such instances brachial birth paralyses are associated. The malformation is discovered during roentgenological examinations or at operation.[62] The dome of the diaphragm forms a sac that bulges into the thorax causing little or no distress. A case observed by me gave no symptoms. Since this baby was delivered by difficult breech extraction one cannot help but wonder if there might not have been an injury to the phrenic nerve in spite of the fact that the brachial plexus was undamaged.

Gastric or duodenal ulcers are probably more common at this age than ordinarily supposed but the diagnosis is rarely made during life. The usual aid to the diagnosis in infancy, e.g., occult blood in the stools, does not have much significance for it is found in 30 to 40 per cent of all newborn. Likewise melena and hematemesis do not necessarily indicate ulcer because this is the time that hemorrhagic disease of the newborn occurs. Perforation of a duodenal ulcer has been reported recently by Somerfield.[63]

Congenital umbilical hernias are rare.[64] In some instances the hernia is so massive that it really amounts to evisceration. These are usually the result of intestinal atresia and are therefore not likely to lend themselves to successful surgical attention.[65] Sometimes the lesion is confused with congenital defects of the abdominal fascia and musculature which occasionally are operable.[66] The smaller hernias are of more practical interest since they may be compatible with life. Spontaneous recovery may occur but in order to avoid peritonitis from necrosis of the cord and exposure of the intestines, the usual complication, surgical attention is indicated. The opening should be closed immediately since

the mortality increases rapidly on delay, owing to the fact that the intestine is relatively sterile in the first few hours after birth.

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9 Exchange Place.

American Academy of Pediatrics

Academy News

The Chairman of Region II of the American Academy of Pediatrics invited the State health officers of the Southern States to meet with the pediatricians of this region for a conference at the meeting of the Southern Medical Association held in Birmingham, November 18, 1932, with the purpose of initiating a cooperative program in the promotion of child health.

As a result of this meeting there was appointed by the conference, three committees as follows:

An Executive Committee, composed of:

Dr. E. C. Mitchell, Chairman
Dr. Horton R. Casparis
Dr. Robert Strong
Dr. E. L. Bishop
Dr. C. W. Garrison
Dr. F. J. Underwood
Dr. L. M. Graves (Consulting)

A Committee of Standards and Practice, or "what to do," composed of:

Dr. Wilburt C. Davison
Dr. William Weston
Dr. Lawrence T. Royster
Dr. James M. Parrott
Dr. Warren F. Draper
Dr. James A. Hayne

A Committee regarding publicity, composed of:

Dr. Luther Holloway
Dr. Alfred A. Walker
Dr. Hugh Leslie Moore
Dr. A. T. McCormack
Dr. J. C. Anderson
Dr. J. N. Baker

On January 5, 1933, the Executive Committee met in Memphis with the following members present: Doctors E. C. Mitchell, E. L. Bishop, Horton R. Casparis, F. J. Underwood and L. M. Graves. The Committee immediately organized by electing Dr. E. C. Mitchell as Chairman. It was decided the function of the Committee was to develop the general outlines and plans of procedure to be followed by the other committees and by such cooperative services as may be developed.

The Committee decided that in developing a cooperative program between public health officials and physicians caring for children, some important age period in child life with which both are concerned should be selected. It is the opinion of the Committee that the period beginning with prenatal life and continuing

through infancy is the most critical in its problems, especially because of the effect on later life. The Committee, therefore, selected this age period for immediate attention.

It is apparent that any program providing for the full application of available knowledge in the protection of the health of childhood must be built around the interest and participation of the practitioners of medicine in the observation, care and supervision of the well child for the reason that without this interest and participation on the part of the physician enduring education of the public is not possible. In other words it is our opinion that we shall never accomplish the best results in the promotion of child health unless the family physician practices preventive medicine and assumes the rôle of adviser and teacher in all matters pertaining to health.

It is the belief of the Committee that a beginning can well be made in this field by encouraging developments under which the local medical society and the local health department will arrange in their communities for the placement of all prenatal cases under the supervision of the family physician with the maintenance of this supervision at least through infancy and with a view of regular periodic observations by the family physician under such financial arrangements as may be made in the particular development.

During the prenatal period, there are certain points which should be emphasized since the welfare of the child is entirely dependent upon the mother's welfare at this time. Examples of these points are: Development and nutrition, congenital syphilis, premature birth, injuries at birth and harmful effects from toxicity of the mother. For the development of activities with reference to this period, the cooperation of the obstetrician is imperative.

Following the prenatal period, it should be recognized that there must be no lapse in the supervision of the child's welfare. Important and immediate objectives should be: A careful and thorough physical examination for the purpose of the discovery of congenital defects or injuries during birth; the institution of measures for the prevention of eye, umbilical, skin, respiratory and venereal infections; the maintenance of adequate fluid intake and normal body temperature; and the careful supervision of both quantity and quality of food including accessory food factors (Vitamin requirements).

With continuation of attention to these objectives, with recognition of the need for transition to a general diet and with emphasis on the need for essential immunizations at the proper age period a basis will have been laid for the reasonably adequate care of the child.

In the attainment of these objectives the Committee recommends the following methods of approach realizing that the entire project is essentially educational in character.

I. Stimulation of the interest of physicians through—

a. Seminars similar in organization and administration to that held annually at Saluda, North Carolina except that it is believed desirable to change the location of seminars developed under this plan from time to time in order that the entire talent available in the South may be utilized and that, as nearly as possible, all interested physicians may be reached.

b. Short-term institutes more local in character and developed with a view of making specialized teaching, pediatric and obstetric talent available to local medical groups.

c. The preparation of printed material having to do with maternal and child care including special handbooks for physicians, diet lists and instructions to mothers for use by individual physicians.

d. Encouragement of medical schools to engage in more extensive post-graduate teaching in the fields of interest here under consideration and in the development in medical schools of "well child clinics" for the objective teaching of undergraduates.

II. Education of the Public.

The acceptance of vital principles, even of the most elementary character, by the general population cannot be secured without the especial and sustained interest of the general practitioner of medicine in his rôle of family physician. In the following enumeration of possible activities, it will be observed that the family physician assumes a prominent position not only in personal contacts but in group work as well. His is the fundamental opportunity, and therefore, the basic responsibility.

Measures which the Committee regards as being of value in education of the public may be summarized as follows:

a. Individual and personal contacts by the family physician, by public health nurses, other public health personnel, specialists, and informed lay people is the foundation upon which any educational program must be erected. Informed public opinion is but the sum of informed individual opinion, hence too great emphasis upon this activity cannot be secured.

b. Lectures either in series or singly to group meetings are useful in reinforcing the efforts of education through personal contact and in conserving the time of special personnel. The most interested groups usually are Parent-Teacher Associations, Women's Clubs, older groups of school children and especially organized study groups, e.g., "Mothers' Clubs." Not infrequently men's civic clubs can be interested in the broader aspects of child care.

c. Special educational measures such as a modification of movements similar in principle to the "Blue Ribbon Program" for school children may be found useful in the stimulation of community interest relative to the care of the early ages of child life. These might take the form of movements calculated to stimulate the interest of older children in the health standards of their younger brothers and sisters thus promoting not only an immediate, but an ultimate objective as well.

d. Miscellaneous mass education measures such as special newspaper articles, printed bulletins, special exhibits and like stimuli of general interest may be found useful in the development of a receptive public attitude and in sustaining public interest.

In order that these methods of approach may be translated into action the Committee recommends the following plan of State organization.

I. A State Committee with a membership may be drawn from either some or all of the following groups:

a. State Medical Association including the councilors.

*b. State Pediatric Society, which should work jointly with State Chairman of American Academy of Pediatrics.

c. The obstetrical group.

d. State Health Department.

e. State Federation of Women's Clubs and/or other like organizations.

f. Medical schools.

It may be found desirable to have an executive committee from this group or even to duplicate the committee organization of the regional body.

*In states with no state pediatric society the regional chairman should assume the responsibility for promotion through the proper channels of the State Medical Society.

II. A local committee in each county, city or groups of counties in which activity is attempted. Here the membership may be drawn from the local elements of the State groups enumerated under the suggested organization of the State Committee since representation of the same interests is essential. Examples are, of course, the County Medical Society, the County Health Department, local women's clubs, et cetera.

Throughout the foregoing discussion the vital importance of the family physician has been repeatedly emphasized. It must, however, be borne in mind that a successful outcome is dependent upon the closeness of cooperation and sympathetic understanding established between the several groups concerned. Each has his part to play, his responsibilities to meet and each should have a profound interest in making the fullest possible contribution to achievement of the real objective, improvement of the standards of child health. True, the family physician assumes a vital rôle, but the public health nurse is here no less than in many other relationships his invaluable handmaiden. The health officer can secure group and individual action that would be unethical for the physician to even attempt. There is, therefore, an opportunity for the service of every group and every individual. There is more work than all together can successfully accomplish.

The tentative program of the third annual meeting of the American Academy of Pediatrics to be held at the Edgewater Beach Hotel, Chicago, June 12, 13, 1933, was announced last month.

On Monday morning, Professor Gorter of Leiden, Netherlands will address the Academy on "Rickets—Dosage of Ultraviolet Light and Vitamin D Determination of Calcium in Bones in Vivo." In the afternoon, Round Table Discussions of various subjects will be held. On Tuesday, there will be a business meeting lasting all day and including the president's annual address.

The following men have been appointed to act on the State Committees:

ILLINOIS

Dr. J. S. McDavid, 715 Lake Street, Oak Park
Dr. Stanley Gibson, 2440 Lakeview Avenue, Chicago
Dr. J. R. Vonachen, 410 Main Street, Peoria

IOWA

Dr. Robert H. McBride, 501 Fifth Street, Sioux City

Comments

DISCUSSION in regard to the various phases of medical economics goes merrily on. We feel that the remarks of one writer to the effect that the report of the five-year study was five years late was one of the most apt criticisms that has been made.

Most physicians will agree that the cost of medical care as paid by the average individual has decreased tremendously in the past two years; further, that the part of the study relating to the income of physicians is in need of a thorough revision.

Much of the discussion recently has centered around the question of some form of "hospital insurance," and many hospital boards and staffs have committees at work studying the subject. In our opinion the agitation for some form of "hospital insurance" is in large part an effort to pull the chestnuts out of the fire for the hospitals. In the flush times of the dim past our hospitals were over built, not only in numbers but in the way of unnecessary elaborateness, which made the cost of maintenance high. It was perfectly smooth sailing when the high-priced rooms were full, income from investments high, and a full public pocket was ready to pay the yearly deficit. Now the situation is different. The expensive rooms are vacant, investment income is fast disappearing, and the annual appeal goes into the wastebasket. And still the hospitals continue to run on with a census of about a 50 per cent capacity. While overhead has been cut, nursing salaries reduced, and food is cheaper, the red figures grow bigger and bigger.

Only a few hospitals have had the courage and intelligence to close or suspend. The common-sense thing would be for many hospitals to combine—temporarily at least—and to keep open only enough hospitals to meet the needs.

Unfortunately many hospitals and their medical staffs cannot see the handwriting on the wall. Not a small percentage of our hospitals have some religious denominational affiliation which makes the matter complicated. We recommend the picture of some of our small towns of a thousand population with five poverty-stricken churches. Even the churches are urging the necessity of combination. Another stumblingblock to combination lies in some one or two powerful board members, often retired from active business, who find in running a hospital a congenial method of "service." The psychiatrist will explain this if the meaning is not clear. A third difficulty lies in the *amour propre* of the chief surgeon or physician (not the pediatrician to the hospital) who will fight to the last ditch to hold his title. And so wallowing deeper and deeper into debt the hospitals will not face the situation frankly and honestly. We leave the hospital superintendent out of the picture, as we can hardly expect him to recommend the doing away with his job.

Like the proverbial drowning man grasping for the straw the hospital managements now turn to "hospital insurance." Under the guise of "better medical care," "spreading the cost," or "better service for the poor" (most of the population) some form of "hospital insurance" is being advocated on every side. In all the discussion we are glad to say we have found one frank and honest hospital executive who admitted his chief interest was in filling the hospital with patients so it could "break even."

There are certain fundamental facts that must be answered in regard to hospital insurance—and so far we have not seen an answer—before we are willing to believe that "hospital insurance" will not lead us further into the mire of present-day medical economics.

Why, when commercial insurance companies have thought out a thousand and one ways of enticing our shekels into their coffers and offering insurance for or against any chance or hazard that exists, have they kept so studiously away from hospital insurance? Even health insurance (cash benefits) are so hedged in with qualifying clauses that the average policy holder is usually out of luck. A number of attempts have been made to furnish "hospital insurance" but all have had a short life. Why is it that hospital insurance is apparently unsound from an actuarial standpoint?

If hospital insurance is to be developed the beneficiaries must come from either one or two sources. Either the insurance must be sold to individuals, or contracts made with organizations. If it is sold to individuals the cost of selling will—it is stated—amount to 25 to 30 per cent of the insurance. The rest goes to the hospitals and physicians (perhaps). If there is only a limited amount of money which today can be spent for medical service, and a quarter goes into business at the start and next the hospitals are put in a balanced financial condition, who, we ask in all sincerity, will hold the bag?

The second method which we have heard advocated—even by members of the medical staff—is for the hospitals to enter into contracts with individual corporations, eliminating the "business cost." Nothing could lead more rapidly, at least in the cities, to a general system of contract medicine without the free choice of a physician.

Why, in the face of all studies and reports showing that the only practical medical insurance is a compulsory one, do the hospitals think they can work out a selected form?

We can only assert it as an opinion, and that's all it's worth, that a large source of the present hospital troubles lies in the excessive number of expensive hospitals which are running at too low a capacity to take care of the overhead. Industry has a solution to overcapacity—it shuts down some of the factories which are not needed to supply the demands and thus eliminates the overhead factor.

This has been done with the hospitals in a few communities and we believe it is a far safer and saner method of attacking the problem than to start on a voyage of "hospital insurance" schemes which are we feel loaded with dynamite.

And now having completed the monthly dissertation on the state of the nation, we can read a few chapters of *British Agent* by Lockhead—the most interesting book we have picked up in many months and go out and work in the vegetable garden with a clear conscience.